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MEDICAL SOCIETY OF LONDON

A SYSTEM OF MEDICINE.

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A

SYSTEM OF MEDICINE.

EDITED BY

J. RUSSELL REYNOLDS, M.D. F.R.S.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON;
FELLOW OF THE IMPERIAL LEOPOLD-CAROLINA ACADEMY OF GERMANY;
FELLOW OF UNIVERSITY COLLEGE, LOND.;
PROFESSOR OF THE PRINCIPLES AND PRACTICE OF MEDICINE IN UNIVERSITY COLLEGE;
PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL;
EXAMINER IN MEDICINE TO THE UNIVERSITY OF LONDON.

VOLUME THE FIRST,

CONTAINING

GENERAL DISEASES.

SECOND EDITION.

London:
MACMILLAN AND CO.
1870.

LONDON:
R. CLAY, SONS, AND TAYLOR, PRINTERS,
BREAD STREET HILL.

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PREFACE TO THE FIRST EDITION.

The object proposed to himself by the Editor of this *System of Medicine* is to present, within as small a compass as is consistent with its practical utility, such an account of all that constitutes both the natural history of disease and the science of pathology as shall be of service in either preventing the occurrence, or detecting the presence, and guiding the treatment of special forms of illness.

As the science and art of Medicine have within the last few years increased very greatly, in regard to both facts and principles, it is held to be desirable,—and indeed almost imperative,—in order to secure the ends that have been mentioned, to divide the large field of growing knowledge into such comparatively small sections as should be enriched by the results of individual and special culture. The Editor feels confident that these results have been obtained; and, in order to secure the like confidence of others, has simply to refer to the names of the many distinguished men who have kindly contributed to this volume.

The general scope of the *System of Medicine,* and the mode in which it has been carried out, render unnecessary any discussion of the so-called "Principles" of Medicine; and the Editor has preferred, by the omission of articles on "general" subjects, to incur the risk of occasional repetition, rather than that of such apparent contradiction as might arise from the treatment of some matters "in the general" by one contributor, and "in detail" by many others.

He has, further, only to express his most grateful thanks to those who have rendered him their invaluable aid; and to submit these results of their toil to the Profession of which they are the distinguished ornaments.

March 21*st*, 1866.

PREFACE TO THE SECOND EDITION.

In this Second Edition, the articles have been submitted for revision to their respective authors. That on Purpura, by the late Dr. Thomas Hillier, has been revised by Dr. Tilbury Fox.

The Editor has introduced into this volume the article on Epidemic Cerebro-Spinal Meningitis, which in the First Edition was placed among the diseases of the Nervous System.

38, Grosvenor Street, Grosvenor Square,
January 18*th*, 1870.

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LIST OF CONTRIBUTORS TO VOL. I.

William Aitken, M.D., L.R.C.S. Edinburgh; Professor of Pathology in the Army Medical School, Netley.

J. Warburton Begbie, M.D., F.R.C.P. Edinburgh; Physician to the Royal Infirmary of Edinburgh.

Hermann Beigel, M.D. Berlin; M.R.C.P. Lond.; Fellow of the Imperial Leopold-Carolina Academy of Germany; Physician to the Metropolitan Free Hospital.

John Syer Bristowe, M.D., F.R.C.P. Lond.; Physician to St. Thomas's Hospital.

Bernard Edward Brodhurst, F.R.C.S. F.L.S., Surgeon to, and Lecturer on Orthopædic Surgery at, St. George's Hospital.

George Buchanan, M.D., F.R.C.P. Lond.; Fellow of University College; formerly Physician to the London Fever Hospital, and to the Hospital for Sick Children; Inspector of Public Health in Medical Department of H.M. Privy Council.

Thomas Buzzard, M.D., M.R.C.P. Lond., University Medical Scholar; Physician to the National Hospital for the Paralysed and Epileptic; formerly on the Staff of H. H. Omar Pacha.

Tilbury Fox, M.D. Lond., M.R.C.P.; University Medical Scholar; Physician to the Department for Skin Diseases in University College Hospital.

Arthur Gamgee, M.D. Edinburgh, late Resident Physician to the Royal Infirmary of Edinburgh.

John Gamgee, Principal of the Albert Veterinary College.

Alfred Baring Garrod, M.D., F.R.S., F.R.C.P. Lond.; Physician to King's College Hospital, and Professor of Materia Medica and Therapeutics in King's College.

Samuel Jones Gee, M.D., M.R.C.P. Lond.; University Medical Scholar; Assistant-Physician to the Hospital for Sick Children, and to St. Bartholomew's Hospital.

Edward Goodeve, M.B., Deputy Inspector-General of Hospitals H.M. Bengal Army; Hon. Physician to the Queen; late Professor of Medicine in the Medical College, and First Physician to the Medical College Hospital, Calcutta; Member of the Senate of the University of Calcutta.

John Harley, M.D., F.R.C.P. Lond., F.L.S.; Hon. Fellow of King's College, London; Assistant-Physician to the London Fever Hospital.

Thomas Hillier, M.D., F.R.C.P. Lond.; Fellow of University College; late Physician to the Hospital for Sick Children, and to the Department for Skin Diseases in University College Hospital.

Jonathan Hutchinson, F.R.C.S. Lond.; Surgeon to the London Hospital, and to the Metropolitan Free Hospital.

John Denis Macdonald, R.N., M.D., F.R.S., Staff-Surgeon to H.M.S. *Victory*.

William Campbell Maclean, M.D. Edin.; Member of the Senate of the University of Madras; Deputy Inspector-General of Hospitals; Professor of Clinical and Military Medicine in the Army Medical School, Netley.

James Furness Marson, F.R.C.S. Lond.; Corresponding Fellow of the Royal Academy of Surgeons of Madrid; Resident Surgeon to the Small-pox and Vaccination Hospital.

Gavin Milroy, M.D. Edinburgh, F.R.C.P. Lond.; Superintendent Medical Inspector to the General Board of Health.

Edmund A. Parkes, M.D., F.R.S., F.R.C.P. Lond.; Emeritus Professor of Clinical Medicine in University College; Member of the General Council of Medical Education; Professor of Hygiene in the Army Medical School, Netley.

John Netten Radcliffe, Hon. Secretary of the Epidemiological Society; Inspector of Public Health in Medical Department of H.M. Privy Council.

J. Russell Reynolds, M.D., F.R.S., F.R.C.P.; Professor of Medicine in University College, Physician to University College Hospital, and Examiner in Medicine to the University of London.

Sydney Ringer, M.D., M.R.C.P. Lond.; Professor of Materia Medica and Therapeutics in University College; Physician to University College Hospital, and to the Hospital for Sick Children.

Edward Cator Seaton, M.D. Edin., M.R.C.P. Lond., Medical Department of H.M. Privy Council.

Edward Smith, M.D., F.R.S., F.R.C.P. Lond.; Inspector to the Poor Law Board; late Assistant-Physician to the Hospital for Consumption, Brompton.

William Squire, L.R.C.P., M.R.C.S. Lond.; late Senior Surgeon to the St. Marylebone Infirmary.

A SYSTEM OF MEDICINE.

INTRODUCTION.

BY THE EDITOR.

DEFINITION OF DISEASE; AND NAMES OF DISEASE.—The attempt to define "disease" must be a failure until we are possessed of a satisfactory definition of "health;" and we are not likely to arrive at this possession until we are able to define the idea that we entertain of the still more fundamental fact of "life." Yet some attempt at definition is not only important, but even essential, for the work set before us in these volumes; inasmuch as the general ideas entertained about disease vary as the years pass on, and the position of "medicine" in the "system of the sciences" is not only expressed by the approximative definition that we frame of disease, but is actually determined by the principle or idea which such definition is constructed to convey.

If we regard disease in "the abstract," we have to deal with that which changes, fetters, renders painful, shortens, or puts an end to life; and, from this point of view, disease may be defined to be any condition of the organism which limits life in either its powers, enjoyments, or duration. We need not stop to discuss the many futile essays that have been made to define that which transcends definition, but which we all, more or less accurately, understand by "life." We accept it as a fact, of which we all know much, but of which we are all assured, by what we do know, that there is much more that we do not know; for it goes beyond our observation, not only at its beginning and at its end, but in its middle term, when it is the most—but even then only partially—exposed to both our senses and our consciousness.

Disease is a condition of the individual man; it is always something more than the changes that we yet can recognise and describe in any particular organ or its function. It is the man who is ill; and, under all circumstances of illness, he has a diminished life. Some organs may be over-active; but their excess of work is needed either because work elsewhere has been left undone, or because it has been done so roughly that parts of organs have been killed before their time, and their wasted materials have to be changed and got rid of with all haste; or because that which held their activity in check has been damaged or destroyed. At all times such overwork is fatiguing and hazardous; often it is directly dangerous, and sometimes it is destructive. Disease is that which limits life—in its usefulness, enjoyments, or duration; and although the body is so constructed that it can often regain its balance when the disturbance has been slight, yet disease, under all circumstances and to all degrees, is the lowering of life, and, even in its most trivial forms, is the "shadow of death."

This mode of regarding disease furnishes us with the *measure* of its importance. A man is ill, or diseased, in degree exactly corresponding with this limitation of his life. A morbid idea may make life miserable and useless; a fatty tumour may be an unfelt excrescence. Many lives are spoiled by diseases that the anatomist cannot name; many lives are but little altered by growths or lesions which may be weighed and demonstrated before a crowd.

If we consider disease from another point, and deal with it as a "fact" of daily experience, we come to regard it as any departure from the structure or functions of the body as these are shown to us in health; and thus we may define it to be an abnormal condition of function, or structure, or both.

But if we contemplate disease in its relation to the many "names" by which its various forms are recognised, we have a complicated problem with which to deal, and can only solve it by endeavouring to separate that which is common to all phases of ill-health, from that which is peculiar to the various names by which those phases may be known, but by which they are only imperfectly expressed. And in order to do this we must recall some typical examples of these names. A patient may be described as suffering from, or he may be said to be an example of, the disease called inflammation of the lung, hooping-cough, tuberculosis, anæmia, typhoid fever, hysteria, or of some other malady, which would or would not readily fall into one or the other of these categories. And be it observed that by this term, "inflammation of the lung," is expressed a particular kind of change in one organ of the body; by "hooping-cough" is meant a special and characteristic variety of a symptom common to many very different affections; by "tuberculosis" is intended some general change in the whole body, distinguished from other general changes by its association with the appearance in one, two, or many organs of a particular material known as tubercle; by "anæmia" is understood literally only an absence or deficiency of blood, but generally an alteration of quality rather than of quantity; by "typhoid fever" is conveyed the idea of a change, of a particular type, in the whole organism, and one which is produced by the introduction into the body of a poison from without; while by "hysteria," and similar phrases, is conveyed some meaning or none at all, and, when the former, a meaning as various in character as are the individuals who use the word. There are other principles upon which disease has been named, and by which it is now described, but these examples are sufficient to show by their very existence the varying prevalence, at different periods, of diverse theories about disease; about the relation of the one organ to the whole system of organs; the nature of the changes which different organs may undergo; the value of particular functional alterations, and of special symptoms; the relation of the blood to life, and to the tissues of the body; the lien between certain materials we can see, and some general conditions we can appreciate by their effect on life; the position in which life-functions stand to the various poisonous agencies around them, and within them; and the concealment, by patent facts of little moment, of important conditions which may be inferred to be their cause. Thus the history of a science might be shown to be written in the names by which the objects about which it is concerned have been described and recognised: bu such is not the end now in view; it is to show that the principles upon which diseases have been named have varied widely, not only at different periods, but at the same; and that so great is the diversity among them, and so strangely aberrant are the forms which disease sometimes assumes, that, hitherto, no self-consistent and at the same time practical nosology has been devised. English physicians have therefore thought it better to retain old names that were well understood, although based upon doubtful, if not erroneous pathology, rather than to invent new terms which could not possess the advantages of their predecessors, although they might very probably share their faults. We have therefore gradually accepted a nosology of most complex composition with the tacit or expressed admission on all hands, that by "pneumonia" is intended much beyond the particular condition of the lung; that it implies changes antecedent to itself in the general nutrition of the body, and alterations in all the tissues and in their processes when the disease itself appears; that the words "hooping-cough" convey more than is included in a particular variety of cough which is characterised by a hooping sound, viz. the well-known history and social relationships of a disease altogether distinct from the paroxysmal cough and hooping sounds not rarely met with in cases of chronic bronchitis and emphysema; that by "tuberculosis" is intended a condition as well marked by general as by local changes, and probably dependent upon some constitutional vice which determines, and is not determined by, the special form of local change; that by "anæmia" is meant much more than the mere etymology of the word can convey; that by "typhoid fever" is intended the description of a disease having relationships only very inadequately expressed by the words in common usage to denote it; and that by "hysteria" and similar expressions are understood more than the present state of medical science will explain, and much more than the words themselves accurately define.

In this state of medical nomenclature, and of medical science as represented by the names it sanctions, it is difficult to arrive at any other *definition* of "disease," than that it is the sum-total of morbid changes in both function and structure; and we must further admit that the *names* by which diseases are recognised are somewhat arbitrary terms used for the purpose of recognition without any constant value as to the meaning of those words in a system of pathology. Sometimes the name expresses what is believed to be the essential or most important fact; sometimes the first link in a long chain of causes and effects; sometimes a characteristic symptom or group of symptoms; sometimes an idea as to the mode of origin of the disease; and sometimes such a negation of all theory, as contents itself with words which shall be understood to mean certain things to which they bear no more pathological relation, although they may have more seeming scientific value, than the common algebraical expressions for "unknown quantities," *x*, *y*, *z*.

But there is, underlying all our nosology, an idea of something special or individual in the diseases that we name. This notion is distinctly expressed with regard to some, hinted at in relation to others, and unsuccessfully concealed in respect of the rest. In spite of opinions to the contrary, the physician knows that there is, in his mind, an ideal type of disease, which he cannot define, and cannot find realized in actual practice, but yet to which type he refers the examples that come before him. Disease to him is something more than a group of symptoms, it is that which makes the group; and he is—as indeed he ought to be—like the naturalist in his process of nomenclature, striving to express, by the names he uses, that which occasions the inner relation between essential facts, and not merely that which will denote the outer and often quite superficial assemblage of phenomena.

As, however, the idea above alluded to is very different in different minds, and pathology is, as yet, in such a condition that it cannot furnish a complete scheme of nosology, based upon the attempt to express, by name, the fundamental fact of all the many maladies with which we are acquainted, disease is defined to be the sum-total of changes from a condition of health which may be recognised in either function, or structure, or both; and the names of diseases are held to be merely convenient expressions for their recognition.

Structural and Functional Disease.—In describing the elements of what we call disease, two terms have been frequently used, "structure" and "function;" and we know that the two phrases "structural disease" and "functional disease" have passed into common usage. Recently it has become somewhat the fashion to object to the latter, and to deny the existence of any such condition. It is necessary therefore to state the grounds upon which the phrase "functional disease" has been retained in this "System of Medicine," and the precise sense in which it is employed.

For this purpose three classes of facts have to be remembered. 1st. That there are some structural alterations, such for example as atheroma in the vessels, which may, if an individual has been killed by an accident, be found extensively distributed throughout the body, the existence of which had been neither known nor suspected by the presentation of any functional change, or symptom, during life. On the other hand, a man may have suffered for many years from discomfort, or marked derangement of the functions of the brain, heart, or lungs, and yet the most practised anatomist, with all means and appliances to help him, may fail to discover, *post mortem*, any organic change which is sufficient to have accounted for them. 2d. Another class of facts, constantly lost sight of by those who deny the existence of functional disease, is to be found in the relations between structure and function in health. At the end of a day's work, and after a night's repose, we might find the two extreme conditions of the organism as regards function. For twelve hours every muscle, nerve, and organ has been doing its utmost, and, as we know, has been wearing out: during the hours of sleep, many organs have been doing little, and some nothing, whereas others have, as it seems, to work on without repose; but in all repair has been going on. By an examination of the body, killed suddenly at the end of one or the other of these periods, it might be possible to infer which had been the condition immediately preceding death. But this inference would be based upon the relation exhibited between the *products* of functional activity, such as the nature, quantity, and quality of the secretions in their several receptacles; and the *raw materials* upon which the organs have to work, such as the nature, quantity, and quality of the chyle, lymph, and blood. It would not be formed upon regard directed only to the condition of organs which had been either in activity or repose. It could not be so based, because the process of repair in the living, healthy body is one that is simultaneous and commensurate with waste. The muscles are not mended up as we mend a damaged wall, by patching up a hole here, and binding on an iron brace or girder yonder; but the process is interstitial; new material is brought in, and brought everywhere; the existing organ is worn down, and the waste matter is carried away; but, with all this change, there is a persistent textural result. Looking at this question still more closely, we see that function is related to structure, not only in the sense that it is what the organ does, but in the much more important meaning that it is at once the expression of the wear and also of the repair of tissues; or in other words the outcome of their life. In the present state of physiology, it is impossible to conceive of a living organ without believing in the nutritive, molecular changes it is undergoing; and these are the essential conditions of its functional activity: it is equally impossible to imagine the function of any living tissue being called into exercise without recognising the dependence of this functional operation upon interstitial movements of repair and waste. But we should be wrong on the other side were we to confound function with the nutritive changes which constitute, not the function itself, but the conditions of its exercise. It is, for example, the peculiar function of a muscle to shorten itself, of a nerve to convey an impulse either of motion or sensation, and of a nerve-centre to convert one of these impulses into the other; the organs referred to, in exercising these functions, undergo certain nutrition-changes; but those molecular changes are not the functions of the organs, but the conditions essential for their performance. This principle, which it seems almost unnecessary to state, in regard of the particular organs or tissues now referred to, is, however, not infrequently lost sight of in respect of secreting organs. It is the function of the salivary gland to secrete a fluid having special characters; of the liver to do this, and to effect changes in the blood which comes to it; and so of other secreting organs: they receive blood into them, and from all of them it passes away, changed; and the organ, as part-product of this change, gives up its secreted matter. These functions, be it observed, depend for their performance upon nutrition-changes in the cells and tissues of the organs; but those fine processes of change are the conditions of functional activity, and are not to be confounded with the thing itself. The secreting cell has to live, to waste, and be repaired; and it lives at a degree of pressure, and is wasted and repaired at a rate, directly proportioned to the amount of work that it accomplishes; and thus it is conditioned precisely as are the ultimate elements of the muscle or the nerve. But minute as is our knowledge of much that goes on in the secreting organs, and of the chemical nature of the results or products of their work, we know no more of the physical conditions which determine that one set of cells shall separate urea, another set saliva, and a third bile acids from the blood, than we do of those which enable one nerve-fibre to convey impressions of light, another of sound, and another those of motion. These are, at present, ultimate facts of physiological science; the function is the expression of the life of the structure; it is what the latter was constructed for the purpose of doing; in doing it, the structure undergoes change; it is wasted and repaired, but these processes are carried on without any breach in the integrity of tissue. Function is to nutrition, as electricity is to the chemic changes in the galvanic battery, a "correlated force." We do not say that the one is the other, but that it is converted into it; and, as in the inorganic world, the arrangement and nature of the particular materials with which different forces come into contact, determine whether chemic action shall appear as heat or magnetism, whether heat shall be shown in motion, light, or electricity, so do the different materials of the living organs, and their arrangement, determine the nature of the functions they perform: how they do this, we do not know, but the facts of physiological science are well known, viz. that the nerve cell exhibits one class of powers, the muscular cell another class, and the secreting organs a third. 3d. A third class of facts to be remembered is, that in many diseases, the only symptoms to be recognised are changes in the degree of activity with which certain organs perform their functions. No new element is introduced by some diseases into the category of vital actions; such affections as chorea, hysteria, epilepsy, might be shown to consist of mere modifications in the degree, time of occurrence, and combinations of functions, each of which, taken *per se*, is consistent with health. The sudden loss of consciousness in epilepsy, for example, is not more mysterious than is the sudden but every-day recurring passage from wakefulness to sleep; the arrested respiration is similar in kind to that seen when the chest is fixed in the performance of any great muscular exertion involving the upper limbs; and still more similar to that which can scarcely be called morbid, the prolonged apnœa of a screaming child, whether the scream be the expression of terror, temper, or pain: the convulsive movements are neither more nor less than nerve and muscular functions, any of which might separately, and many of which might in combination, be the expression of healthy vital activity.

From these three classes of facts, therefore, we are compelled to admit that, in the present state of science, the *onus probandi* lies with those who assert the constant presence of structural in association with functional change; and we affirm that those who make the assertion have never proved their point. Further, that as a matter of inference from what we know of the relation subsisting between structure and function in health, we should not even expect to find solutions of continuity or coarse changes of texture in those diseases, the essential elements of which are functions altered, not in kind, but only in degree and mode of association; and that, on the other hand, when we do find material changes in association with functional disturbances, we should refer many of the latter only indirectly to what we see of the former, the more numerous and more important of them being dependent upon what we do not see, viz. the finer changes in the interstitial processes of nutrition. A scirrhous tumour of the stomach, for example, may produce certain symptoms easily explicable by its mechanical effects; it may be so situated as to prevent the ingress or egress of food; but vomiting may occur when the orifices are free, or when the tumour is situated in some organ in the pelvis: the supposed tumour may cut off the supply of food, and so explain some of the changes we see in colour and general nutrition; but, on the other hand, the extreme of wasting and of cancerous tinting may be seen when there is no such enforced abstinence from food, but when indeed a large quantity is not only taken, but is digested and enjoyed. So again a clot of blood in the corpus striatum may sever the nerve-fibres, and so explain the severance between the will and certain muscles of the extremities; but it will not so explain the presence of convulsions, or of spasms in those palsied limbs.

For these reasons we retain the words "functional disease;" understanding by them such changes as have no recognised morbid anatomy, but such as depend upon corresponding changes in the finer processes of nutrition. We do not believe that there is any altered function without a correlated change in the nutrition of the organ; but what we assert is that such change, as a matter of fact, is of such kind as to be undiscoverable by our senses, and as a matter of inference, from what we know of the relation between nutrition and function, is of such nature that it may always be beyond the reach of observation. No healthy function is performed without nutrition-change: no morbid function can exist without altered nutrition-change; but the relation between the two elements, "structure" and "function," is the same in the two conditions.

Nothing is more erroneous than the common notion that "functional" means trivial, and that "structural" means severe. Many diseases, designated by the former word, are long-continued, obstinate, or destructive; many known by the latter are of short duration, are amenable to treatment, or are harmless. Diseases which spoil or shorten life are not trivial because they depend upon such fine changes as may escape our observation; but they are the more serious when they thus elude our notice, just because they have their place in the very centre—the most ultimate process and fact—of life, the conduct of nutrition.

NATURAL HISTORY OF DISEASE.—Under this phrase are recorded the symptoms or phenomena of disease, their causes, the manner of their development, their duration, and the different modes of their termination, whether the termination be in death, or in a return to health. In the natural history of disease no theory is involved; we have to deal only with facts.

Causes.—Commencing with that which precedes the appearance of symptoms, viz. the "causes" of disease, there are some principles which it is important to lay down; inasmuch as our ideas of the causation of disease are determined, in great measure, by our conceptions of what disease itself is. Nothing is more easy sometimes than the discovery of a cause, and its distinct separation from a particular and well-known malady. For example, a healthy child is brought into a room where some one is suffering from scarlet-fever, and after a certain period it exhibits symptoms of the same malady, and passes through all its stages. Here we say there was direct communication of the malady; but we must not forget two facts, first that some children so exposed do not take the fever, although they have not previously had the disease; and secondly, that those who have suffered from it once rarely take it again, although they may be exposed to infection. We suppose a constitutional disposition in one case, an indisposition in a second, and assert the existence of the latter in a third. Still, when the cause has operated we feel that we tread on safe ground when asserting broadly that the cause is "infection."

Nothing, however, is more difficult in some cases than to say what the cause of a disease has been. For example, six people take an indigestible meal, and one of them suffers nothing, a second is troubled with dyspepsia, a third with asthma, a fourth has an epileptic fit, a fifth an attack of gout, and a sixth is disturbed with diarrhœa. One element in the causation of all these maladies is the same, viz. an indigestible meal, but the results vary widely; and we say that this is owing to constitutional conditions which "predispose" to these particular affections. These have been called "predisposing causes;" but we must inquire what they are, and how they are related to these diseases, if we would understand the latter. Among the predisposing causes are reckoned hereditary taint, sex, age, and constitutional peculiarities, either congenital or acquired; and with regard to two of these, hereditary taint and constitutional peculiarity, we must admit that they are, in reality, disease. When we allow the existence of either, we but throw back a few steps further the line, and widen the circle which includes all that we mean by the disease itself: we include in the malady more than its name expresses; we partially account for its occurrence, but do not explain its "cause." It is quite true that we may sometimes draw a line between certain so-called causes and effects; we may say, for example, this man, of tuberculous family and with latent tubercle, was yesterday apparently well, but he was exposed to cold, and to-day he has tubercular pneumonia; his constitutional state "predisposed" him to the evil, which the exposure "excited" into activity; but regarding him from a pathological point of view in his present position, that of a sufferer from tubercular pneumonia, we cannot separate the elements of his disease so easily, for it was not a cause of his malady which was there before, but an integral part of the affection under which he is now labouring. A similar difficulty is to be encountered on almost every hand; the worry of the Stock Exchange is borne by some men bravely, others succumb, but variously, one goes mad, another is "broken down," a third becomes epileptic, and so on; and we fly to the resource of "predisposition," some weakness somewhere, which this wear and tear has pointed out, and urged into morbid activity; but in that very weakness, if there was not the whole of the disease called A, B or C, there was some important element of it, and not its remote or predisposing "cause." If disease be, as we believe it must be, defined to be the sum-total of changes in either structure or function, or both, then almost all of these so-called "predisposing causes" are part of the disease itself.

But those other conditions, sex and age, it may be asked, are they integral elements of disease, or are they its cause? *Sex* cannot be said, accurately, to be a cause of disease any more than the same can be affirmed of life, but yet, in all modern treatises on medicine, it figures in the chapter on ætiology. Be it observed, however, that the word "sex," when thus used, has reference to many conditions of difference beyond those which exist in the reproductive organs. The male sex, as a rule, exhibits the masculine frame of body and of mind, while the female sex displays the opposite; and their respective "predispositions" to disease have relation to these characters as well as to the physical structure of the apparatus of reproduction. The existence of an organ is the necessary condition of its becoming diseased,—a man cannot suffer from ovarian tumour, nor a woman from orchitis,—but there are some organs common to the two sexes, and equally developed in each until the sexual distinctions of puberty are seen; then in the one they become rudimentary and inactive, whereas in the other they take on new and important functions. In these instances sex may be said to be a predisposing condition of disease, but it is so only in this sense, that it exposes certain organs to some of the causes of disease by the simple fact of their functional activity. It must not be supposed that the healthy action of any organ predisposes it to morbid change; on the contrary, it exerts rather a strengthening and protective influence; but such activity simply lays it open to the operation of influences which cannot be brought to bear upon its counterpart which remains in a rudimentary condition in the other sex. But besides these essential differences there are others which are accidental, and which depend upon education, practice, and habits of life, as these are determined by the customs, fashions, or peculiarities of the people and their times. And, further, there are many facts with regard to the relative proclivity of the sexes to special diseases which have not yet been explained by any known relation of these maladies to the reproductive organs. Simple ulcer of the stomach and carcinoma of the same organ afford examples of this difference of proclivity, and we at present have to regard them as ultimate facts of Pathology.

Among the conditions which determine, therefore, the differences of sexual predisposition to disease, we reckon (1) the actual presence or absence of the organs; (2) the action or almost absolute inaction of the organs; (3) the relation between the amount of activity of some functions of certain systems of organs, and other functions of the same systems, where differences exist *ab initio;* and (4) the effects of habit, education, and fashion, in either producing or lessening functional activity, with all its correlated structural conditions.

In a similar manner must we regard the influence of *age* in the production of disease. It is not *per se* a predisposing cause, but it carries with it certain things which may be. If we can conceive of a perfectly healthy organism, placed in absolutely healthy conditions, then we may believe that it would pass through the stages of growth, dentition, puberty, adult life, and decline; and that it would perform all the functions of self-preservation and reproduction without either hurt or hindrance; sometimes, nay, very often, we do actually see some of these stages passed through with as entire a freedom from discomfort as any ideal being placed in the most Utopian circumstances could wish for; but at some point or another in the long course of life, the chain of good succession is broken by a faulty link or an unexpectedly heavy blow, and then follow one or another of the many ills that make up the miseries of common life, and average health. On the other hand we see cases in which nothing seems to be capable of going well: every epoch, every change, every organ, seems, as it were, pounced upon at every turn by all the evil agencies that surround it; action or inaction, growth or decay—it matters not which—seems exaggerated into a condition of disease, and life is a lifelong misery. It is clear that no one period of life is in itself a cause of disease, or of exemption rom its occurrence, since all periods may be passed through without ny disturbance of the health, and no period possesses absolute mmunity from its attack. But it is also evident that the changes vhich take place at certain periods, render some individuals liable to he operation of other causes, and that this operation may be very ffectual in the production of disease. Such periods are those of rapid tructural development, and the commencement of new functional ctivities, or the decline of structure and the arrest of action. That vhich would seem to be the condition tending to morbid development s the disturbance of the balance of activity and growth between lifferent organs, or systems of organs, so that for a time at least an ndue preponderance is given to a certain set. Thus, during the first lentition, although there is general growth, there is particular activity f certain organs; and, in like manner, at the commencement of uberty, there is, as it were, undue prominence given to the organs ffecting reproduction; and although the particular structures involved n these developmental changes may not be selected as the localities or morbid action, the very fact of their disproportionate activity—by listurbing the balance of general nutritive progression—may become cause of derangement in other systems of organs, such as the vascular r nervous. In like manner, at the climacteric period, the repression f certain functions, to the operation of which the whole body has ecome habituated through a long series of years, may prove itself the tarting-point of morbid changes in the functions of other organs vhich are, by this repression, placed in new and trying circumstances. y these considerations we may, for the most part, explain the nfluence, where it is marked to the highest degree, of age as a o-called "predisposing cause" of disease.

But there are some maladies which appear to have a definite elation to age, and which are not explicable upon these principles; nd with regard to these we must admit that certain periods of life are ssociated with proclivity to disturbances of particular kinds, the ature of the relation between the two classes of conditions being at resent unrecognised by medical science. At the same time the istory of all science is such that it teaches us to believe that these elations will be hereafter discovered and found to be analogous to hose which we already understand and appreciate.

The term "exciting cause" of disease has been applied to another lass of condition altogether, and one with regard to which there is much less difficulty. That which is involved in the term is the peration of some influence from without, be that such as to act upon mind, emotion, sensation, nutrition, vascular conditions, temperature, r any other function or property of the living body. A great surprise, or an overwork, may affect the mind; a domestic calamity, r the worry of business, may disturb the emotional centres; a physical injury may set up changes in sensation; a forced rest may weaken muscular nutrition; a tight bandage, or an altered position, may influence the vascular supply; cold or heat may diminish or increase the temperature of parts; and in like manner other agencies may affect the organism, and become the exciting causes of disease. The form which the latter take is determined partly by the nature of the exciting cause, and its relation to this or that system of organs; and partly by the condition of the organism, in regard of hereditarily received or congenitally acquired morbid state, the peculiarities of sex, and the period of life.

Two things therefore concur to make up what we term the causes of disease, but they are essentially distinct, and it would be better to denote them by different names, than by one name differently qualified. The one, the so-called "predisposing cause," is a "diseased condition," and is therefore a part of the disease itself; the other has no necessary relation to the individual or his constitution, and is in reality a "cause" of the disease from which he is suffering.

Symptoms of Disease.—The meaning which now we must assign to the word "symptom" or "sign" of disease, is very different from that which some time ago would be conveyed by those terms. So long as disease was regarded as something put into, added to, or engrafted upon the body; a material, or other entity,—having even a more or less substantive existence,—these words described the means by which we might recognise the presence of such an entity: but so soon as disease is recognised to be, as we have defined it, the sum of changes in function and structure presented by the living being, the words "symptom" and "sign" have another meaning, and describe only those parts of the disease which are appreciable by others. Disease is a complex state of a complicated organism, and, although the *name* which we may give it may be intended to express its primary or most important fact, we cannot separate this one fact from others with which it is associated, but must regard them as integral parts of the malady we have either to study or to treat. They may differ, from an outside point of view, in proximity of relationship; but the heat of skin, the altered pulse-respiration ratio, the nature of the expectoration, the changes in the secretions, in the nervous system, and in the prospects of life, together with the altered resonance, breath and voice-sounds, are as much parts of the disease called "pneumonia" as are the structural conditions of the lung. Some of them may be signs by which we recognise its presence; but they are also essential elements of the malady itself. In like manner it might be shown with regard to those other diseases, the nomenclature of which differs as we have already described, that a precisely similar relation exists between what we have denominated "symptoms" and what we understand by "disease." We cannot know of the existence, during life, of any disease except by its symptoms; we cannot conceive of disease apart from some recognisable changes in either function or structure; and these changes constitute the disease: nor can we, on the other hand, imagine the existence of what we call "symptoms" apart from the correlative idea of what we conceive to be "disease." The two classes of notion have been, of necessity, distinct in their development;

but the maintenance of the distinction between them has been a hindrance to true progress in pathology; and it will be well for us to try and remove that hindrance. So long as "disease" is thought of as a something—it matters not what—distinct from the "phenomena," or "symptoms," by which it makes itself known, so long are we in danger of mistaking its real meaning, and of overlooking those true guides towards the removal or alleviation of its evil, an end to which all medical science ultimately points.

"Disease," we have said, is a change of structure, or of function, or of both; "symptoms" are those changes in structure, or function, or both, which we can recognise. The latter, the symptoms, are not separate from, but are parts of the disease, and their only characteristic is that they are such parts as are appreciable during life. But they differ in kind and in value among themselves, and have been known by different names, so that we meet with such distinctions as those between "signs" and "symptoms," between "objective" and "subjective" symptoms; and between "general" and "local" symptoms. These terms almost explain themselves, and are retained because they possess a certain amount of utility; but it is more easy to make use of them in practice than to define their exact meaning in the abstract. The idea underlying the word "sign" is that it is some physical change which can be observed directly; and thus we speak of dulness on percussion, tubular breathing, augmented vocal fremitus, and bronchophony, as "physical signs" of condensation of the lung; whereas we speak of dyspnœa, expectoration and fever as "symptoms" of pneumonia, or tuberculosis. But heat of skin, an eruption on its surface, the wasting of a muscle, or the fact and quality of a secretion, are as much "physical signs" of a disease as are the particular phenomena we have mentioned. Yet it is almost unknown, it is certainly very unusual, to hear the term "physical sign" applied to any of them. It is easy to trace the origin of this distinction between symptoms and signs to the period when the physical examination of the chest arrived at its due position. It was felt that an amount of precision in diagnosis was arrived at by percussion and auscultation, such as was never dreamed of in the times before such modes of exploration were employed; and therefore the conditions revealed by their aid were expressed in terms differing from those which had been previously employed, to describe such changes as short-breathing, pain, expectoration, and the like. But it is impossible to maintain the distinction; the number of respirations per minute is as much a "sign" as is the dulness on percussion, and both of them may be "symptoms" of disease. Everything that may be observed is both the one and the other, and the reason why the distinction has been maintained is, because with regard to the former—the "sign"—there may be but one means for its recognition, namely, observation from without; whereas with regard to the latter—the "symptom"—there is a possibility of confounding two things essentially distinct, namely, the sensations of the patient, and the phenomena he presents. Deficiency of resonance is discovered only by percussion; but short-breathing is a sensation of which the patient complains, as well as a phenomenon which may be appreciated and measured by the physician; and in like manner wasting of a limb may make itself at once evident to the observer, whereas paralysis is a condition which the patient may assume.

The really valuable element of distinction between these two classes of phenomena is therefore that which exists between "objective" and "subjective" symptoms; meaning, by the former word, all those elements of disease which can be appreciated by the senses of the observer, and by the latter, those which can only be known through the statements of the patient. Whatever of disease comes to us only through the mind or feelings of the patient, as expressed by language, either of gesture or words, is a "subjective" symptom. Its form and its degree of intensity are subjected to the conditions of the machinery—mental, emotional, and sensational—through which it passes, and it is liable therefore to be changed, either in character or degree. Whatever of disease comes to us through our own senses alone escapes this danger of addition, alteration, or subtraction, and is *pro tanto* of higher value. In one class the patient gives his version of his case; the symptoms are such as we can only get at through his mind, and they are termed "subjective:" in the other, we make our own observations—the feelings or ideas of the sufferer have nothing to do with them—simply, physical facts are the objects we recognise, and such symptoms are called "objective."

As a general rule the objective are much more valuable than the subjective symptoms; but let it be remembered that the importance of the latter is very widely variable, and that sometimes it may far exceed anything that can be derived from direct observation. In the early stages of some serious diseases of the heart, or brain, nothing may be presented to the practised ear or eye; and yet the patient tells us of a deep unrest, or sudden horror, which, although it has no objective sign, may be the herald of a sudden or lingering disease; as true and as important—although to others the mind seems clear, and the heart's beat healthy—as any murmur we might hear with the stethoscope, or any palsy we might measure by the hand. We have to deal with man as a whole; and to ignore or undervalue what he tells us of his ideas, emotions, or sensations, because they may be termed "subjective symptoms," and be held to be therefore unreliable, would be to shut out from ourselves that which—egotistic and fearful, prejudiced and ignorant as man may be—yet forms an integral part of his life, and therefore of his disease. We must be careful to give to both groups of symptoms their true value, and our danger in the present day is to underrate the importance of those which, a few years ago, constituted almost the total symptomatology of disease.

The distinction between "general" and "local" symptoms need not detain us, since the terms are obvious in their meaning, and the difference between them is gradually dying out by the recognition of the fact, that no one organ can have its functions or its structure changed without the existence of some relative change in all the rest.

Course.—In describing some few diseases we have little more to do than to detail the phenomena present at any one given time; whereas in furnishing the natural history of others we are compelled to speak of premonitory symptoms, or prodromata, of the modes of commencement of the illnesses, their forms of attack or their *débût*, and of the different stages—two, three, or more—through which they pass. Between these two extremes we have every amount of variation: the natural history of one disease may be compressed into a sentence; that of another may expand into a book; but more commonly we have to deal with histories intermediate in duration, and perhaps less emphatic in their interest.

It often happens, however, that the mode in which one event follows another is of great importance in the diagnosis of disease. For example, the decumbency in pleurisy has different meanings at the commencement of the malady, and at its later stages; and in like manner rigid muscles teach one thing at the onset of a paralytic seizure, another when the immediate effects are passing off and the patient is regaining power; and still a third when without such restoration it makes its appearance at a yet later period, and in a well-known order.

Not merely the mode of sequence, but the actual time of sequence is of diagnostic and therapeutic value; and this we know full well in the study and recognition of the acute specific diseases, of malarial fevers, and the like; and with equal significance, though with less accuracy of measurement, can we use the element of time in the diagnosis of many chronic diseases; and such element may sometimes be sufficient to determine a question left wholly in the dark by other elements which we have had before us. In this manner time enters occasionally into the diagnosis of tubercle, of cancer, of hæmorrhage, and of other maladies; and it would be difficult to overrate its value in those special cases which are left in obscurity by the absence of special or pathognomonic symptoms.

Again, the relative intensity of symptoms is a point to which attention must be frequently directed, for by this alone a diagnosis may be possible. If we regarded the relative intensity of pain, dyspnœa, fever, cough, and general nutrition-change, each of which might be present in bronchitis, pneumonia, pleurisy, phthisis, or asthma, it might be possible to say which disease existed, and this without the aid of auscultation or percussion. And in like manner by regarding the relative amount of coma, paralysis, spasm, and rigidity, it would be possible to distinguish, in some cases, between cerebral hæmorrhage, acute softening of the brain, congestion of the brain, or urinæmia.

All these particulars have to be described under the head "course" of symptoms; and they form an essential part of the natural history of disease.

Duration.—The importance of a consideration of time in the diagnosis of some classes of disease has already been referred to; it is of no less importance when, diagnosis being determined, a knowledge of the natural history of disease gives us power to foretell, with some approximation to accuracy, its probable results.

We know approximately the duration of herpes zoster, of vaccine, or variolous pustules, of typhus, or scarlet fever; and we know also approximately, the duration of phthisis pulmonalis, of carcinoma ventriculi, and of other maladies. With regard to the latter—the chronic class—our knowledge is much less definite, or rather it lies within a larger range, and is of less practical utility. It may, regarded from a pathological point of view, be even more accurate than that which we boast of in respect of the acute diseases; for the relative range of variation is not greater, although in the one case we deal with days or even hours, and in the other with months or years. But life is on the side of the balance, and death is on the other, and the balance is struck between those two, whether the beam be long or short; and so we congratulate ourselves upon a readily used and readily appreciated forecasting of the immediate, present evil, and perhaps underrate our sounder knowledge, with regard to that which may not happen for ten, twenty, or a hundred months.

The duration of symptoms, therefore, though of variable social value, is of vast interest in the natural history of disease.

Termination.—A disease may end in various ways:—(1) The patient may gradually get rid of it altogether. (2) He may lose all the urgent symptoms—all that at one time seemed to constitute the malady—and yet retain some less urgent symptoms; or some which are not recognised at all; or some which—so far as the patient's own feelings are concerned—are not, even after recovery, recognisable, but which may be discovered by a physical examination of the organs instituted by the physician. (3) He may continue to present all the local disturbances, while the general or constitutional changes pass away, and thus the disease persists, but in an altered and what is called a "chronic" form; or (4) the disease may become worse, pass from one stage of weakness to another, and end by the destruction of the patient, which may be brought about in various ways. Now, all these points in the natural history of disease, are of much interest in regard of prognosis, and of treatment. The recognition of early signs of recovery is a great help towards the choice between therapeutic means, and is a good ground for employing one of the strongest of these means of cure, viz. hope. The search for and discovery of the effects of an acute illness—although in that acute form it has passed away—are of great importance to the life, prospects, work and career of the unconscious sufferer. We must know these probable consequences if we would perform our duties as the advisers of those who place themselves under our care. It is not enough to guide a disease to an apparently successful issue; we must know what weak point it may have left behind, and we must guard this with the utmost caution. Again, the tendency to pass into a chronic form often reveals the existence of some constitutional vice we had not before suspected, and thus renders itself available for a more effective direction of our therapeutic agencies. And, lastly, the perception of the early indications of a fatal issue may be of great social or individual value; whereas the recognition of the mode in which death threatens to approach, may be the means of teaching us to select such measures as shall temporarily, or even indefinitely, postpone the evil. Thus the "terminations" of disease, whether they be in health, in impaired health, or in death, are not only of interest to the natural historian and the pathologist, but are full of teaching to him whose aim is to render natural history and pathology subservient to the great work of healing diseases, of relieving them, or of measuring their duration, and lessening the pain with which they do their work.

DIAGNOSIS OF DISEASE.—In the earlier days of medical science, the problem of diagnosis might have been stated thus:—"given the symptoms, to find the disease;" but, in these days, such problem must be translated into the following terms:—"given some of the elements of disease, to discover the others." We do not now regard eruptions on the skin, peculiar changes in the mucous membrane of the throat, an elevated temperature, and a disturbed innervation as the signs by which some morbid "entity" in the body reveals its presence; but as parts of the morbid condition of the organism, from which we may infer the existence of simultaneous changes in stomach, intestines, liver, spleen, or kidneys. The rose rash of typhoid is as much a part of the disease as is the ulceration of Peyer's glands; the dulness on percussion, the altered condition of the urine, the changed nervous power, are as truly elements of the disease called pneumonia as are the minute alterations which constitute inflammation of the lung. Some of the elements of disease escape our observation during life; some are directly perceived by us; and others are appreciated indirectly by such processes as percussion, chemical examination, laryngoscopy, spirometry, and the like: but the only difference between these is their relation to the observer; they occupy precisely the same position in regard to the disease itself. The process of diagnosis therefore is the passage, not from effects to causes, not from phenomena to noumena, for, strictly speaking, we do not at all know what are either the causes or the noumena of disease; but diagnosis is the process by which, perceiving some particulars, we infer the existence of others, which we know to be commonly associated with them. This is however the pathologic or scientific side of diagnosis; and the question may still be asked, What is it practically? Practically, diagnosis is the process by which to a certain set of symptoms we affix a more or less familiar name; and become able to say that such an one is suffering from typhoid fever, meningitis, hooping-cough, epilepsy, or gout. And when we have given some names to diseases, we have, more or less intentionally and with differing degrees of accuracy, conveyed some theory into the process of diagnosis. Remembering what was said with regard to "names of disease," we shall find that, in this practical sense, diagnosis is sometimes the discovery of and the calling of a disease by what we conceive to be the most important structural change by which it is accompanied, such as pneumonia; at other times diagnosis stops short at the recognition of a pathognomonic symptom, such as hooping-cough: again, it may mean to express the starting-point or principal fact in a group of symptoms; or some general condition but imperfectly understood, or some condition which is not understood at all. In all of these instances we notice two things: first, that diagnosis—looked at from its scientific side—is the step from particulars to particulars, from those which are observed to those which during life can only be inferred; the step from the one or the one hundred to the whole; second, that diagnosis—regarded practically—is the giving of a name to the disease from which an individual suffers; the ticketing, and as it were placing of that malady in some niche with others that resemble it, so that it may be known and brought out when required. But let it be remembered that the principles upon which names are constructed are so various that no expression can be framed to convey their meaning, in so far forth as that it should carry further what is meant by diagnosis.

PATHOLOGY.—When a patient presents himself for examination or for treatment, he tells us of certain things that he feels or sees, which are wrong, and which we call "symptoms;" we ask him questions, and learn additional facts of a similar class; we observe him, and notice other facts which he can neither feel nor know by independent means, and these we call "signs," or "objective symptoms." From what we are informed, and from what we observe, we pass on to the inference of other facts: we believe, when some three, four, or more changes from the healthy state are present, that other alterations exist; and this belief is more or less strong, and its character more or less definite, according to the state of medical science, and our knowledge of it, at the time that the patient presents himself. We *infer* certain things, certain conditions which we cannot see, but which we may, in some measure, classify, and therefore call by distinctive names. Thus heat of skin is a symptom from which we infer a number of ulterior conditions. The reason for this heat of skin may—other symptoms being regarded—be conjectured by us to be an altered blood-state, such as typhoid fever, tuberculosis, or the like: it may, on other symptomatic evidence, be referred to a change in some particular organ, such as inflammation of the lung, or destruction of a portion of the spinal cord: and we may give names to the general conditions, part of which we have observed, part of which we have inferred. This is what we call "diagnosis" in its practical sense: but that which makes diagnosis possible is the existence of a certain amount of information about the relations subsisting between different organs, and about the modes in which particular organs may become altered in function or in structure. We observe a change in the quantity, colour, or other qualities of a certain secretion, and we infer from this that there is diminished, increased, or perverted action of a certain organ. We examine further, we discover other changes, and we infer the nature of the disease which that organ has undergone, or through which it is now passing. What in this sense is true of one organ is, more or less, true of all, and of the organism as a whole, so that we are able to give some general expression to its general state; and thus we speak of paralysis, fever, anæmia, suppression of urine, weakness, &c. &c., and so advance from symptoms—by diagnosis—to pathology. This is what we do in particular cases; finding certain symptoms, and knowing certain principles, we place the individual who presents those symptoms in a particular category, and call his disease by a name that is understood: and in this way the process of thought which we call "diagnosis" is a bridge across from "symptoms" to "pathology." Diagnosis is, then, the practical application of what we know about pathology; and its accuracy will be in proportion to the amount of our information, and to the keenness and readiness of our observing power. Pathology, therefore, is the foundation, the essential condition of diagnosis: for it would be simply impossible to advance, from the observation of symptoms, one step towards the recognition of the nature of disease, unless that step were based upon pathology; *i.e.* upon a knowledge of the conditions under which morbid changes occur in structure and in function, and also of the modes in which such changes may so affect organs as to render themselves appreciable during life. Practically, as cases come before us, we observe symptoms, and we diagnosticate pathological conditions; but we could form no diagnosis without pathologic knowledge, and diagnosis is in reality but a portion of, or the practical application of pathology. Theoretically, however, pathology is the groundwork of diagnosis; and not only so, but of all valuable and correct observation: and thus the one is seen to be but the necessary complement of the other in the practice of physic. The one is a science, the other is an art: the latter could not exist without the former; the former grows daily by the application of the latter.

By Pathology, then, we mean the general doctrine of disease, the knowledge of the conditions under which it occurs, and of the kind of change which it expresses in the functions of the body. We mean also the inter-relations of different organs, or systems of organs, whether these may be compensative, or sympathetic, or antagonistic of one another. By the pathology of a disease we mean the scientific classification and nomenclature of its phenomena, and the interpretation of the conditions under which they have arisen. Pathology is to the body, under diseased conditions, what physiology is to the healthy organism, viz. the law of its being, or rather the best expression that we can give to what we believe that law to be. In its detail it must embrace all changes in either structure or in function; but in its common and general acceptation it is used to express the idea that is entertained of the primary or essential or most important change in both; and the bearing that this has directly upon life as a whole is, in its statement, the unravelling of the problem of what we ordinarily mean by "Pathology."

Pathological Anatomy.—Structural changes in some organs are so constantly and definitely related to functional alterations observed during life that "Pathological Anatomy" in some maladies constitutes the most important, if not the major, part of their pathology. There are other cases in which structure-changes cannot be shown so to account for all the detail of symptoms; and in these, pathological anatomy has its interest, but is of less immediate value. And further, there are diseases with regard to which pathological anatomy has hitherto taught nothing that is worth our knowing; while, on the other hand, it has not seldom exhibited the existence of disease, never suspected during life, and yet dire enough to be the cause of death, and thus to show that our diagnostic powers are not such, that we may boast of them.

Unquestioned as is the value of Pathological Anatomy, it is no less unquestionable that this value may be overrated and misapplied; and it is possible that such mistake may exist at the present day. Let us, therefore, remember what Pathological Anatomy is, and what it teaches. It is the condition of an organ after death, when tissues are cold, ordinary chemic changes have begun, secretion and living motion have ceased, circulation has stopped, and all that we know to be unexplained by ordinary physical conditions, and that we call "vital," has gone, and gone we know not whither, and know not how. Pathology, on the other hand, refers to the conditions and modes of action of the organs when they are warm; when chemic changes seem to be directed by a power that cannot yet be explained by chemic force; when sensation and motion are constantly placing the organs in new positions, in regard to other organs, and in new conditions, in so far as relates to their own integral parts; when the blood is constantly flowing, and affecting, as it does so, both the tissues and itself, not leaving them as it found them, not leaving them as it came; when all that we understand, and all that we dimly guess at about that complex force or principle, combination of forces, condition or what-not, that we call "life," is the director or harmonizer of all we see, or is engaged in what seems a fierce struggle with powers it has long directed, but which, having now risen in defiance of its authority, seem likely to accomplish its dethronement.

Everything that we can see is altered by that change from life to death; and Pathological Anatomy has to do directly with physical conditions, which may be partially causes, partially effects, partially conditions of, and partially unrelated to the disease of which they are but a part, although an important one. If everything that we can see is thus altered in the step that all bodies make before they come under the scalpel or the microscope of the pathologist, still more altered is almost everything that we cannot see, but which we have inferred to exist from our laborious physiological investigations. It would seem therefore that Pathological Anatomy is about as capable of furnishing, *per se*, a notion of disease as dissection would be of teaching physiology; and if we are to exalt into such undue prominence—as it has been the fashion of late years to do—this branch of science, which is yet only a branch and not the science itself, it would be nothing more than consistent to attempt to write biographies from post-mortem examination and to construct the history of a nation by exhuming the bodies from its graveyards.

Pathological Anatomy has reference to one element of disease—structural or tissue change—and that only under conditions very different from those which exist during life, and when only disease can be said to exist. Further, we get the materials for examination when disease has passed through all its stages, and through that final one, which cannot be said to form a part of either life or of disease. It is only when, by accident or intercurrent malady, a patient is struck down in the earlier stages of morbid change, that we have the opportunity for observing the kind of alteration which exists at such most important periods; and even to them is added all that we have already described, and probably much more than we can even guess at, by the great fact of death. Disease, being a complex of change in function and in structure, is represented anatomically only by the latter: disease, having a history, and passing through stages, is shown to us only in one stage, and with infinite predominance of frequency in the last stage: disease, being a change in the conditions of life, is shown to us anatomically only in the condition of death: while General Pathology, therefore, may embrace all that we know or can know of the mechanism of human suffering, Pathological Anatomy but touches the human body when the period of suffering has passed.

But the facts revealed by a study of Pathological Anatomy have a peculiar kind and a high degree of value, which we would most unwillingly underrate. There is an intimate and constant relation between function and structure (see p. 5); and we believe that although certain structural conditions have yet escaped our observation, they may hereafter be rendered patent to the senses of the observer; and, on the other hand, we hold that there are some structural changes which may always transcend our powers for their immediate recognition: but, notwithstanding these facts and principles, we must remember that where anatomical changes have been discovered and verified, they have thrown unexpected light upon previously dark and complicated problems; and that they have possessed—in themselves and by their very nature—a definiteness, or precision, and have been susceptible of description and measurement to a degree that is quite unattainable by our present modes of research in regard of functional change. In Pathological Anatomy, what we have, however little it may be, is definite, describable, demonstrable, and measurable. These characters constitute its great value; and it is against its undue exaltation—and not its proper use—that we raise our protest. Let us be as exact in our inferences from its facts as we may be in our observation and description of them, and then Pathological Anatomy will take its proper place, and we shall learn from it its most useful lessons.

Prognosis.—The practical test of a true science is the power which it confers of "prevision," or of knowing now what will follow hereafter. Some sciences have attained to this point, as we see daily illustrated by physics and chemistry; but as yet medical science has arrived at only very partial security of forecast. And yet the foreknowledge of the consequences of a present disease is that for which patients and their friends often seek from the physician with the greatest eagerness. When we can prognosticate with certainty, medicine will have become a "science." At present we only, with different degrees of nearness, approach this end. We may describe the "probabilities" of a given disease; we may even measure them; we may accept or reject lives at insurance offices; or we may affix a numerical value to their duration;[1] but we deal with doubts, and not with certainties. Life is too subtle for us to know or measure all its possible contingencies; and our information is too scanty to render us thoroughly satisfactory interpreters of the outcome of any malady. But, with all this doubt, much may be accomplished for the safety of society, and the relief of individual anxiety or care.

In prognosis we have almost always two ends to be considered: the immediate effects of the present illness, and its remote consequences upon life. We have also two main elements by which we are guided in judging of these ends, viz.: the local changes which we may directly appreciate, and the general conditions which we may infer to underlie them, or to be, in some way, their consequence.

Prognosis with regard to the immediate effects of a present malady is guided mainly by the degree to which it interferes with any or all of the great vital functions, the circulation, respiration, innervation, or nutrition of the body. Prognosis in regard of the ultimate issue of a malady is based upon all these, but still more frequently upon the recognition of changes, often minute, in either function or structure, which our knowledge of pathology leads us to interpret for either good or evil. For example, a man is suffering from pneumonia, and we may—finding his general functions performed with an amount of ease consistent with life—infer that he will recover from the attack which to other eyes may appear most threatening; but we may discover physical signs of tubercular disease—slight, even unnoticed previously—and these teach us that the ultimate prognosis is unfavourable. In like manner, a child may be taken with convulsions which appear as frightful as only convulsions can do, and yet we may give a favourable opinion as to the present illness; while, on the other hand, some much slighter convulsive movement accompanied by heat of skin, a variable pulse, an obstinate vomiting and constipation, and a history of failing health, or of hereditary tubercular taint, may lead us, in the midst of what seems a trifling malady, to augur the worst results from what we believe to be the onset of tubercular meningitis.

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[1] See p. 16, on the duration and termination of diseases.

In prognosis, therefore, we must bear in mind the two objects we have set before us, and the two classes of means by which we may advance towards them. The immediate prognosis turns upon the degree to which great vital functions are interfered with: the remote depends upon the nature of slighter changes, of which pathology teaches us the meaning.

THERAPEUTICS AND HYGIENICS.—In the prevention or treatment of a disease our science culminates and becomes an art. Unless it can accomplish one or the other of these ends, the world would do as well without as with our aid. It is of some value to know the probabilities of our state, but it is of comparatively small value to have this knowledge if we can do nothing either to ward off, alleviate, or cure disease. We may prepare some people for the worst, we may dispel some groundless fears; but our mission is to do more than this: we have to try to "cure the curable, and comfort the incurable."

In the prevention of disease regard is had to the condition in which the individual is placed, to his hereditary or acquired constitutional peculiarities, and to the minute physical or functional departures from health which we may discover. In the treatment of disease no one of these can be lost sight of with impunity, but we are guided principally by the actual symptoms present at the time. These symptoms, however, it must be remembered, are of two orders: from the one set we learn the actual physical condition of an organ, or a group of organs, such as inflammation of a lung, congestion of the liver, or paralysis of one side of the body; while from the other series we become acquainted with the state of the system generally, whether this be antecedent to the local change, its cause, its effect, or a mere coincidence of its existence; and from such a group of symptoms we infer the existence of dyscrasiæ, such as tuberculosis, carcinoma, or the like, and appreciate the presence of vigour, or of asthenia, of sound constitution, or of impaired health and wasted strength.

To the most superficial observer it must be obvious that therapeutics has undergone great and important changes, that the mode of treatment now adopted for many diseases is just the opposite of that which was in vogue a generation ago, and which lingers even in recent editions of standard books, although their authors have long since ceased to follow the directions which they still give to others. A few years ago the treatment of inflammation of an important organ was laid down definitely; such and such things were to be done, and no questions were to be asked as to whether the case was of this, that, or the other type. Inflammation was there, and blood was to be taken; low diet was to be enjoined, and lowering medicines were to be exhibited; and supposing the inflammation did not yield, the forces of attack were to be again placed in action: but here evidently there crept in some distrust of the theory at the bottom of the practice; for, instead of general bleeding, leeches or cupping were to be employed, and then only to a mild degree. Somehow or another the inflammation was to be put down, and it not rarely happened that the process urged against the bugbear "inflammation" proved fatal or highly injurious to the patient. If we can, by bleeding, and by it alone, save the eyesight which may be threatened by iritis, or if we can by depletion save a life which is endangered by laryngitis, we are quite justified in adopting that measure, although it may entail some injurious consequences. It would, however, be as unkind as it would be unphilosophical to relieve the pain of a simple pleurisy by abstracting blood, in such amount as should damage the individual in after years, when equal relief might be obtained by poultices and patience. We still find it written, if these conditions are found—a hard, full, strong, frequent pulse, with great heat of skin, no prostration, impending evil from this condition being patent as the phenomena themselves—then bleeding, antiphlogistics, and the like must be employed. But, as a matter of fact, we do not find these cases, and the more common *on dit* of medical practice is to the effect that as the inflammation seemed extending, the quantity of wine has been doubled, the supplies of beef-tea increased, and bark and ammonia given more frequently. Partly to account for, and partly to justify, so material a change in our modes of dealing with disease, it has been assumed that the *vis vitiæ* of the British constitution has been lessened, or that the so-called "type" of its maladies has altered; an assumption which has little to be said in its defence, and still less that can be regarded as its establishment. A more simple, and we believe accurate, explanation of the change is to be found in this, that previously theory was the groundwork of therapeutics, and that now fact is the basis of treatment: that, years ago, diseases were treated by their names, and that now they are treated by their known conditions: that local changes were the main guides in times gone by, but that the general state of the patient is that which in these days the physician esteems as his therapeutical informant. When pathology scarcely existed, medical practice was an empirical art; and had, with the few advantages of that position, all its evils: whereas, with the growth of pathology, therapeutics, still an art, has become, or is becoming, a science; and, knowing more accurately the limits of its powers, is content to attempt less heroic measures, being convinced that it does less harm. Much is done by medical treatment now, more real good than ever was done before, but it is done in a different way, and with another aim. Disease is detected in its earlier stages, and often arrested there; and when developed the patient is guided through it, if he can be, and is not sacrificed to some wild attempt at its destruction.

What we now believe and act upon is no set theory regarding the nature of particular diseases, or disease in general—modern times have not been devoid of theories upon which the fathers of medicine would justly have turned their backs in derision; but such notions, although they may have misguided a few individuals, have soon found their proper place, or no place at all in the science of the day —what we do believe and act upon is a better knowledge of the laws and relations of morbid change: we see that the man is greater than his maladies; that his general condition is of more importance than his local ailments; that disease is a change in him rather than in some part of him; and that no treatment can be of any real service which sacrifices the greater to the less. In all treatment therefore what is general is to be dealt with upon the basis of a true appreciation of the general pathological condition, and this in spite of all theories in regard of local changes, however they may be termed, whether they come to us with names hoary with age, or scarcely intelligible, and even sometimes ludicrous, from their novelty. If the general condition be one of weakness, it matters not that the brain, the heart, or the lungs may be in the state of so-called "inflammation;" the weakness is the one thing that demands immediate treatment, and to neglect its treatment is to run the risk of sacrificing the patient to a theory of a compound state even now but imperfectly understood. This is the starting-point, the essential element in therapeutics; but the mode in which the treatment should be applied will often be determined by the nature and position and origin of the special lesion; and these conditions of the latter will direct the management of those means and appliances which, employed locally, will prove of service to the injured organ.

CLASSIFICATION OF DISEASES.—A correct classification is a condition of the existence of a science, and an essential for its teaching; but we do not think that "medicine" has yet arrived at this high position. That it will advance to it, that it is making progress towards it, we have no doubt; but, at the present time, we must admit that imperfections abound in every system that has been propounded. The problem is too vast and too complicated for solution now, and we have therefore to adopt that which appears to possess the greatest amount of practical advantage.

It would be useless here to spend time upon criticism of the various schemes which have been proposed; we prefer rather to state briefly the very simple plan upon which this book will be constructed.

It is proposed to make the first division of diseases into their two great groups; 1st. Those in which the whole organism appears primarily and prominently deranged, and 2d, those in which special organs or systems of organs are, in like manner, affected. Subdividing the first group we have two classes: A, those in which the disease appears to be developed by causes operating from outside the body; and B, those in which the malady seems to depend upon some internal change. Thus in the first subdivision we find the acute specific diseases, and their analogous affections; in the second gout, rheumatism, scorbutus, and the like. Subdividing the second group we have many classes, consisting of diseases of systems of organs, such as: A, diseases of the nervous system; B, diseases of the digestive system and its appendages; C, diseases of the circulatory system; D, diseases of the respiratory system; E, diseases of the urinary system; F, diseases of the reproductive system; G, diseases of the locomotive system; and H, diseases of the cutaneous system.

Each of these is, in its turn, again subdivided, upon the primary principle of general or partial change, so that, in regard of the nervous system, for example, we have 1st, those of general or undetermined seat, and 2d, those depending upon distinct local change in its parts, anatomically considered: and this leads to further reduction into affection of parts, such as, *a*, brain; *b*, spine; *c*, nerves: whereas the final division is based upon the nature of the changes which these portions of systems undergo.

If this mode of arranging diseases has no other merit, it has that of simplicity; and it will, we believe, bring, as a general rule, into closer proximity than some more ambitious systems would allow, those diseases which have the most intimate clinical association. It involves little theory in any case, none in many, and may therefore commend itself to those who realize, amid the great progressive science of medicine, the difficulties and dangers which attend upon all nosologies which, based on theories, partly right and partly wrong, carry with them, and only with great effort disentangle themselves from, what is erroneous in their groundwork, and *à fortiori* luxuriant in their after-growth.

J. RUSSELL REYNOLDS.

PART I.

In the First Part of this System of Medicine are included those diseases in which the whole organism is primarily and prominently deranged. We have, therefore, to deal with:—

General Diseases, or Affections of the Whole System; *and, dividing these into two sections, have to consider first:—*

§ I.—*Those determined by agents operating from without, such as malarial diseases, the exanthemata, and their allies.*

Influenza.
Hooping-Cough.
Croup.
Diphtheria.
Scarlet Fever.
Dengue.
Roseola.
Measles.
Mumps.
Sudamina and Miliaria.
Varicella.
Variola.
Vaccination.
Glanders.
Hydrophobia.
Enteric Fever.
Typhus Fever.
Relapsing Fever.
Yellow Fever.
Epidemic Cerebro-Spinal Meningitis.
Plague.
Erysipelas.
Pyæmia.
Malarial Fevers.
Dysentery.
Diarrhœa.
Cholera.
Syphilis.

INFLUENZA.

By Edmund A. Parkes, M.D. F.R.S.

Definition.—An epidemic specific fever, with special and early implication of the naso-laryngo-bronchial mucous membrane; duration definite of from four to eight days; one attack not preservative in future epidemics.

Synonyms.—*Scientific Names.*—Peripneumonia Notha (*Sydenham*, *Boerhaave*). Peripneumonia Catarrhalis (*Huxham*). Pleuritis Humida (*Stoll*). Febris Catarrhalis (*F. Hoffman*, *Sauvages*). Catarrhe Pulmonaire (*Pinel*). Catarrhus à Contagio (*Cullen*). Defluxio Catarrhalis. Cephalalgia Contagiosa. Rheuma Epidemicum.

Popular Names.—Pose (in old English writings, from the Anglo-Saxon *gepose*, heaviness). Tac or Horion (in France in 1411). Coqueluche (in France in 1414, and in subsequent epidemics, because the sick wore a cap over their heads). La Dando, or Ladendo (in France, 1427). Quinte (in France in 1578, because the paroxysms of cough returned every five hours). Follette (in France in several epidemics). Ziep (in Germany in 1580, probably from *zieppen*, to pipe or chirp). Schaffhusten and Schaffkrankheit (in Germany in 1580, because the cough was like the cough of a sheep, or because the vertigo was like the sudden giddiness of sheep). Hühner-weh (in Germany in 1580, because the cough was like the crowing of a cock). Blitz-katarrh (from the suddenness of the attack). Mal del Castrone (in Italy in 1580, because the giddiness was like the common disease "turnsick," of the sheep).

In the seventeenth century it was first called Influenza, in Italy, because it was attributed to the "influence" of the stars, and this term has passed into medical use.

In 1743, it was called La Grippe in France, from the Polish Grypka (Raucedo) a term which, like Influenza, has passed into medical writings.

It has been called in Russia "Chinese Catarrh;" in Germany and Italy, "the Russian disease;" in France, "Italian Fever," &c.

A great number of other popular names have been given to it:—Petite poste; Petit courier; Follette; Coquette; Cocote; Allure; Baraquette; Générale, &c.

History.—Supposed to be referred to by Hippocrates, who yet gives no perfect description. The epidemic among the Athenian army in Sicily (415 before Christ), recorded by Diodorus Siculus (lib. xiii.), has been supposed to have been Influenza. In

A.D. 827 an attack of cough, which spread like the plague, was recorded. Again, in 876, Italy, and then the whole of Europe,[1] was attacked, and the army of Charlemagne, returning from Italy, suffered greatly;[2] dogs and birds were both attacked by this disease. In 976 the whole of France and Germany was attacked by a fever, whose principal symptom was a cough. In 1173 another catarrhal epidemic was widely spread; and in 1239 and 1299 other slighter epidemics are noticed.

It is not, however, till the 14th century that the records became numerous and precise:—

In the 14th	century	6	epidemics are recorded.
15th	„	7	„
16th	„	11	„
17th	„	16	„
18th	„	18	„
19th	(first half)	10	„

In some cases, however, the same epidemics may have been recorded twice, though I have excluded several that appear to have been so. Probably, also, among the lesser epidemics are some of hooping-cough wrongly diagnosed as Influenza.

There is little doubt that the apparent increase of prevalence in the last centuries is merely due to more accurate recording of minor epidemics.

Of these epidemics, some have been very widely spread over a great part of the known world, as in 1311, 1557, 1580, 1590, 1729, 1762, 1775, 1780–2, 1830–2, 1847. In other epidemics the disease has either been partial, or not recorded in many places; in some instances it has spread only over comparatively small tracts of country. The first epidemic which was carefully described was that in 1557, by Riverius. The great epidemic of 1580 was described by Sennert.

In England the following epidemics have been recorded,[3] many of them with great care:—1510 and 1557 by Thomas Short; 1658 by Willis; 1675 by Sydenham; 1729—1743 by Huxham; 1732–3 by Arbuthnot; 1758 by Whytt; 1762 by Baker and Rutty; 1767 by Heberden; 1775 by Fothergill, who collected observations from many physicians; 1782 by Gray, Haygath, and Carmichael Smith; 1803 by Pearson and Falconer, and a great number of others; 1833 by Hingeston and others; 1837 by Streeten, Graves, and Bryson, &c.; 1847 by Peacock,[4] Laycock, and many others.[5]

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[1] Schnurrer. Chronik der Seuchen, Band i. p. 175.

[2] Schnurrer states that measles followed, and appeared indeed to be developed out of this epidemic, but the records are necessarily very imperfect.

[3] Annals of Influenza, by Theophilus Thomson (Sydenham Society, 1852). I have only quoted the principal works.

[4] The Influenza, or Epidemic Catarrhal Fever of 1847-8. By G. T. B. Peacock, M.D. London, 1848.

[5] Since 1847 there has been no pandemic Influenza, but several minor outbreaks, the widest diffusion of which was in 1857-58. In Paris in 1867 (February and March) there

SPREAD OF THE DISEASE.—*Etiology.*—Before entering on the consideration of the external nature of the causes, it is necessary to state the facts which have been ascertained in respect of the spread of Influenza.

It has prevailed in most places of the habitable globe: in the whole of Europe; in China, Tartary, Egypt, India, and other parts of Asia; in Australia, Polynesia; in North and South America, and in the West Indies; that is to say, in both hemispheres and in all latitudes. It has occasionally occurred in both hemispheres at the same time, but more usually has appeared successively in different places, and has been seen at some point or other of the earth's surface for two to four years, after which it has disappeared. In some years, as in 1580, 1730, 1762, 1775, its prevalence has been so great, that almost all parts of the known world have been attacked; at other times it has been more partial, affecting only a part of a continent, or even a single country.[1] It has been supposed, indeed, occasionally to prevail in quite a limited area, in a single city for example,[2] but it is possible that local catarrhal fevers of this kind are not identical with the true Influenza.

When it has been pandemic, *i.e.* when it has invaded a large portion of the earth's surface, its progress has usually been rapid, yet not to such an extent as is commonly supposed, and sometimes it has travelled slowly. It is said to have overspread Europe in six weeks, and at another time to have taken six months or more to do so. In any particular country its progress may also be comparatively slow: thus, between the invasion of London, and of provincial towns, or of Scotland, weeks, and even sometimes months, have elapsed. Thus in 1762 it appeared in London in the beginning of April; at Edinburgh in the beginning of May; in some parts of Cumberland in June. In 1782 it attacked London in the middle of May; Exeter at the end of May; and Newcastle-upon-Tyne and Edinburgh in the beginning of June.

In 1830–31–32, it prevailed in Moscow and St. Petersburg, and from thence took no less than eight months to spread over the whole of Germany. In Europe it has sometimes prevailed simultaneously at several points, as in 1847, when it was raging at the same time in Copenhagen, London, and Marseilles. In spreading over a large tract of country, it has often been supposed to follow a regular course; which has been believed to be from the high north or north-east to the south and west. Thus it has been supposed to pass from Chinese Tartary to Russia, Germany, Holland, England, Scotland, France, and then to Italy and the Mediterranean, or to America in succession, and certainly in some epidemics there has been a course of this kind. But this is by no means invariable, and may indeed have been accidental, or our knowledge of the successive steps of the spread

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was a severe outbreak, characterised as usual by great prostration. There was also an outbreak almost at the same time at Strasburg.

[1] See Hirsch, Hist. Geog. Pathol. vol. i. p. 286, for twenty-four examples of this fact.

[2] Hirsch has collected seventeen examples.

may have been inaccurate. Thus the epidemic of 1762 was said to follow this course,[1] and to reach America in October 1762, having affected Germany in February and March, London in April, and France in July. But Influenza prevailed in America the year before (1761) and thence passed into Europe,[2] taking thus the exact contrary of the tract assigned to it, unless indeed it passed round by the icy regions of North British and Russian America, of which there is no evidence. So again it has sometimes (1775) passed from the south to the north of Europe, or from the south or west of a particular country to the north or east. Gluge,[3] indeed, from an examination of the epidemics of the last 300 years, believed he had discovered that its course is from west to east. It is obvious that, in former times, the want of reliable information, and of intercourse between nations, must have rendered all evidence of dates very uncertain. The next epidemic will give more reliable information than any of the former.

When it has entered any large town, it remains there for from six weeks to two months as a rule, but occasionally longer, as at Paris in 1831, when it was more or less prevalent for nine or ten months. It has never, however, failed entirely to disappear eventually, and sporadic cases are not seen in the intervals of the epidemics. In its course it appears to pass over seas, and it is said to have attacked the crews of ships far from land, who had not sailed from an infected port.[4]

The exact spot on the earth's surface where an epidemic commences has not yet been made out, and two opinions prevail. One is, that every epidemic owns one unknown source, whence it spreads; each nation, in turn, attributing to its neighbour from whom it derived the disease, the unenviable honour of originating it. Thus the Italians have termed it the German disease; the Germans, the Russian pest; the Russians, the Chinese Catarrh; and these names are indeed some evidence of its usual track. Noah Webster attributed its origin to America in 1698, 1757, 1761, and 1781, while in 1788 he believed it arose in Europe, and several writers have fixed it in Chinese Tartary, or in India. The other opinion is, that it has no special place of origin, but may arise anywhere; and some,[1] indeed, have questioned whether such "autochthonic" developments are not the rule, and whether we are right in believing in a "genetic connexion" of the various local outbreaks. But surely no one can doubt the connexion of the various attacks in the great epidemics of Influenza with some general and pandemic influence.

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[1] Robert Williams. On Morbid Poisons (1841), vol. ii. p. 663.

[2] Noah Webster. A Brief History of Epidemic and Pestilential Diseases (London, 1800), vol. ii. p. 44.

[3] Quoted by Hirsch, op. cit. p. 287, footnote.

[4] The statement that Influenza will thus break out in mid-sea, without there being any possibility of the disease having been introduced on board, is a most important piece of evidence, as it would prove that the atmosphere can not only carry the poison, but that no degree of dilution can destroy it. Without denying the occurrence of such outbreaks, I cannot but consider we require better evidence of ships being attacked in mid-ocean. In some of the quoted instances, the ship had been at a port either known to be infected, or in which Influenza was really present, though it had not become epidemic. As we are ignorant of the exact period of incubation, some men may have been infected before sailing. In other cases the examples are of old date, and it is impossible to feel quite sure that the evidence is correct. Such for example as the celebrated case of the *Atlas* East Indiaman, which was attacked with Influenza, on a voyage from Malacca to Canton; Malacca being healthy at the time, but Canton being affected at the same time as the ship. (Robert Williams: On Morbid Poisons, vol. ii. p. 667.) In 1782, Admiral Kempenfeldt's and Lord Howe's squadron, cruising at different parts of the Channel, were each attacked, although, it is said, they had been at least twenty-two to twenty-seven days at sea.

There is better evidence that ships near the land have suffered. In 1833 the *Stag* frigate was coming up Channel, and when off Beechy Head, in Devonshire, the wind was easterly and off the shore at two o'clock, the crew being then quite healthy (and it is presumed no communication having taken place, but this is not stated)—forty men were at half-past two, suddenly attacked with Influenza; at six o'clock sixty men were down and by the next day 160. (Watson: Principles and Practice of Medicine, 4th Edition, vol. ii. p. 44.) If it were certain that there had been no communication with the shore, the cause must have drifted over the sea.

If it may arise thus spontaneously in various places, no one has yet precisely indicated its first origin.

It has been also supposed to have a cyclical course, and to return pretty regularly in periods of years. The older writers thought it had a cycle of about 100 years, but it has returned much more frequently than this; about every twenty-five to thirty-five years it has been pandemic, and lesser outbreaks have occurred more frequently. But no regular period can be at present perceived.

In passing through a country it does not attack all parts of it; it more usually spares the country places, but sometimes even large towns escape.

When the disease enters a town, it has occasionally attacked numbers of the inhabitants almost simultaneously. But more frequently its course is somewhat slower; it attacks a few families first, and then in a few days rapidly spreads; the accounts of thousands of persons being at once attacked at the onset of the disease are chiefly taken from the older records, in which the suddenness of the outbreak is exaggerated. Frequently, perhaps always, in a great city the outbreak is made up by a number of localized attacks, certain streets or districts being more affected than others, or being for a time solely affected, and in this way it successively passes to different parts of the city. It has generally occurred in a great city before appearing in the smaller towns and villages round it, and sometimes these towns, though in the neighbourhood, have not been invaded for some weeks.

In some cases, and perhaps a large number, it breaks out after persons ill with Influenza have arrived from infected places.

The decline in any great town is less rapid than its rise, and usually occupies from four to six weeks, or sometimes longer.

In every epidemic the symptoms so closely resemble each other that there is no difficulty in recognising it from the descriptions even of ancient and unlearned writers; yet there are said to be certain slight differences in symptoms between different epidemics, to which reference will be hereafter made.

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[1] Biermer, in Virchow's Handbuch der Path. und Ther. vol. v. p. 607. (1865.)

Different epidemics have varied somewhat in the number of persons they affect, but on the whole a large number suffer.

In London in the last epidemic (1847) it has been calculated that at least 250,000 persons suffered; in Paris, between one-fourth and one-half of the population suffered, and in Geneva not less than one-third.[1]

When the different telluric and atmospheric conditions which are coincident with the attacks are considered, the following are the conclusions :—

Soil.—It prevails on every soil and geological formation, and apparently equally on all. It has been supposed to be worst on marshy soils, and some have even believed it to arise in very malarious regions, as Lower Bengal, or the plains of China, yet very malarious countries, as Holland, do not suffer more than others ; Holland, indeed, has escaped some epidemics which have traversed Europe. Lowlands have been sometimes affected more than the adjacent hills, as in the lowlands in Jamaica, in 1802, and in several epidemics in the Lombard plains, as compared with the Blue Mountains and the Alps.

Volcanic Eruptions.—Telluric Emanations.—Noah Webster[2] and Schnurrer[3] have collected the available evidence on this point, but it is entirely negative. There have been constant volcanic eruptions without Influenza, and epidemics of Influenza without great volcanic eruptions. It has been thought that emanations of seleniuretted hydrogen from volcanoes might excite Influenza, but no proof has ever been given of the existence of this substance in the atmosphere.

Electrical Conditions.—No evidence has been collected which shows any connexion with conditions of telluric magnetism or atmospheric electricity; and indeed the peculiar spread and frequent localization of Influenza seem inconsistent with general magnetic conditions.

Seasons.—The disease appears at all times of the year;[4] nor is there any reason for considering it an affection of the late summer, autumn, and winter, as has been stated.

Temperature of the Air.—Owing to the confusion in the popular mind between Influenza and common catarrhs or catarrhal fevers, it has been always a common opinion that Influenza depends either on a low temperature, or a sudden variation of temperature. This error has taken a long time to kill; but almost every writer, since the epidemic of 1580, has examined this point,[5] and has decided that there is no connexion between either low temperature, or variations in temperature, and Influenza. As respects high temperature, it has prevailed in the West Indies at a temperature of 72°—82°[1]; on the hot sea-coast of Java; in South India; in Egypt; at the Cape of Good Hope, in the most genial season; in the South of Europe in summer. So also there is abundant evidence to show that the changes of weather, which may appear to have accompanied or preceded its outbreak, were mere coincidences.[2]

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[1] Peacock on Influenza, p. 13, Introduction.

[2] A Brief History of Epidemic and Pestilential Diseases. 1800. Vols. i. and ii.

[3] Chronik der Seuchen. 1825.

[4] See Hirsch, op. cit. p. 287, for evidence on this point, but almost all writers have noticed it.

[5] Salius Diversus (1580); Molineux (1693); Whytt (1757); Baker (1762); Haygarth (1775—1782); Fothergill (1775); Metzger (1800); Lombard (1831), &c.

Moisture of the Air.—It has prevailed in the dry air of Upper Egypt; in the moist air of sea-coasts, and on the sea itself, without being apparently in any way influenced.

Barometrical Condition.—No coincident alteration can be traced.

Ozone.—Although ozone was known before 1847, the observations during that year led to no result, and since that time there has been no epidemic of Influenza. But the observations hitherto made on the effect of ozone on other diseases[3] seem to render it improbable that any connexion will be traced between the development of ozone and Influenza. The statements of Schönbein are based merely on the effect of large quantities of ozone artificially produced, on the mucous membrane of the nose. Applied in large quantities, ozone is irritating, and may produce simple catarrh, but nothing like the specific symptoms of Influenza.

Fogs and Mists.—In some cases, as in Paris in 1675, France in 1733 and 1775, England in 1782, a thick and acrid fog has shortly preceded, or has immediately ushered in, the Influenza; but so many outbreaks have occurred without such a coincidence, that it is impossible to attach any weight to it.

Wind.—Its main spread is not influenced by the wind; it does not move with the same velocity; it often moves against it. Yet it appears to be sometimes carried by the wind for a short distance. (See case of the *Stag* frigate, previously quoted.)

In fine, if there is any special atmospheric condition which invariably attends epidemics of Influenza, it has yet to be discovered, and the words of Pearson are still true:—

"Between the epidemic and the condition of the atmosphere, there appears to be a connexion different from that which depends on a mere alteration of temperature, or of dryness or moisture, but what that peculiar connexion is we shall not attempt to explain."[4]

Fungi in Atmosphere.—Abundance of flies, caterpillars, &c.—Attempts have been made to show that during epidemics there are indications of an unusual development of animal or vegetable life, and that "bloody or red snow," "blood rain," "flights of locusts, or insects," &c. are more common in Influenza years. These speculations have, at present, even more than usual interest, and certainly should be brought to the test of close inquiry. At present, all that can be said is that no facts of any moment exist which connect an unusual fungoid development with the spread of Influenza.

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[1] Observations relative to the West Indian Islands, by John Williamson, M.D. 1817. Vol. ii. p. 110.

[2] In his late work (Catarrh and Influenza, 1865), Seitz attributes more influence to the effect of vicissitudes of weather in causing epidemic Influenza than appears to me to be warranted by the facts.

[3] Especially those of Shiefferdecker: Sitzungsbericht der Math. Naturw. Classe der Wien. Akad. July 1855, Band xvii. Seite 191. The ozonic results had no connexion with any malady, and were in all cases proportionate to a numerical range, derived from a consideration of the strength of the wind and of the moisture of the air. See also Seitz (Catarrh and Influenza, 1865, p. 360).

[4] Observations on the present Catarrhal Fever, or Influenza, by Richard Pearson M.D. London, 1803. P. 3, footnote.

We must now pass on to a different order of facts.

Human Intercourse.[1]—The rapidity of the spread would seem at once to negative any connexion between human intercourse and the propagation of the disease; yet there is some affirmative evidence. It does not appear to follow the great lines of commerce; but when it has entered towns and villages in which the investigation can be carried on, it is curious how frequently the first cases have been introduced, and how often the townspeople nearest the invalids have been first affected. In this country especially, Haygarth in 1775 and 1782, and Falconer in 1802, collected so many instances of this, that they became convinced that its propagation was due entirely to human intercourse.[2] So also, when it passes through a house, it occasionally attacks one person after another. But if it is introduced in this way, it afterwards develops with marvellous rapidity, for we cannot discredit the accounts of many thousand persons being attacked within a day or two, which is quite different from the comparatively slow spread of the contagious diseases. This *sudden* invasion of a community makes it, to many persons, appear highly improbable that any effluvia passing off from the sick should thus so rapidly contaminate the atmosphere of a whole town.

Still, we must remember how singularly, of late years, the knowledge of the introduction of cholera by persons coming from infected districts has increased, and how very striking are the instances of this kind already recorded in several works on Influenza.

In some cases, again, isolation or seclusion of a community, as in prisons, has given immunity; or at least that community has not been attacked.

Inoculability.—The disease is not inoculable; at least, when horses are attacked, it cannot be transferred from one horse to another. (Hertwig.)

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[1] The presumed importation of Influenza into Iceland and the Faroe Islands, as described, especially recently, by Schleissner and Panum, as well as by older writers, is doubtful. It would seem probable that the endemic catarrh of these islands, said to follow each year the arrival of the first ship, is different from the true Influenza, which comes more rarely, and only when it is prevailing elsewhere in Europe.

[2] Sir Thomas Watson, M.D., whose care and accuracy inspire such faith, says also on this point:—"The instances are very numerous, too numerous to be attributed to mere chance, in which the complaint has first broken out in those particular houses of a town at which travellers have arrived from infected places." (Principles and Practice of Medicine, vol. ii. p. 43. 4th Edition.)

Sir George Baker was one of the first who noticed this fact. (Opuscula Medica: Edition of 1814, p. 27.)

Cullen's term, "Catarrhus à Contagio," seems to me to imply, however, merely the idea of origin from a special virus.

Incubative Period.—All the contagions have one remarkable property; there is a time when they are said to lie dormant, and to be undergoing or inducing in parts of the body those changes which lead at last to the symptoms of the declared disease. During this period there are either no symptoms, or, what is more probable, they have not been determined.

Such a period has been supposed not to exist in Influenza,[1] which has been said to strike down persons in perfect health, as with a stroke of lightning. But the suddenness does not exclude an incubative period without subjective symptoms. It is also certain that the incubative period sometimes exists. It is sometimes very short;[2] sometimes of many days' duration.

Preservation from Second Attack.—There is some discrepancy of evidence; but, on the whole, it seems clear that, while persons seldom have a second attack in the same epidemic (though even this may occur), an attack in one does not protect against a subsequent epidemic. Indeed, it has been supposed rather to render the body more liable.

Relation to other Epidemic Diseases of Man.—It has been attempted to trace out a connexion between Influenza and measles, the plague, yellow fever,[3] and cynanche maligna (diphtheria). It has been supposed also to precede and herald cholera. On the other hand, it has been stated that epidemic scarlet fever disappeared when Influenza prevailed, and reappeared when this ceased. The same fact has been affirmed of small-pox.[4] During its prevalence other severe inflammatory diseases have been supposed to lessen. With regard to all these supposed relations, the evidence is most unsatisfactory. Coincidences between the prevalence of different epidemic diseases must be expected, but it would require repeated instances to prove any connexion. Measles constantly prevail without Influenza; and if an epidemic has occasionally followed an Influenza epidemic, this really proves nothing. The utter want of connexion between cholera and Influenza is evident at a glance.

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[1] Biermer, op. cit.: "The disease seems to come on without an incubative stage; the causes of influenza do not work after many days, as a contagion; but rapidly, like a poison."—P. 604.

[2] In the Transactions of the College of Physicians (vol. iii.), it is stated that in the epidemic of 1782, seventeen persons came to London to an hotel, and on the following day three were attacked with Influenza. Haygarth (On the Manner in which the Influenza of 1775 and 1782 spread by Contagion in Chester and its Neighbourhood, by John Haygarth, M.D. F.R.S.) says that a gentleman came to Chester from London, on the 24th of May, 1782, ill of Influenza; a lady, into whose family he came, was seized on the 26th, and was the first case in the town. Haygarth states, evidently with the wish to point out the possibility of a direct contagion, that the gentleman was engaged to be, and was afterwards, married to this lady. In this case the longest possible incubative period was two days. In 1782 a family landed at Harwich, from Portugal, and came to London directly; the day after their arrival, the lady, two servants, and two children were all seized. Two men-of-war arrived at Gravesend from the West Indies; three Custom-house officers went on board: a few hours afterwards the crews of both vessels were attacked. (Robert Williams, on Morbid Poisons, vol. ii.) Some other cases are on record where the incubative period, if it existed, could not have been more than a single day. On the other hand, some cases are on record in which the incubative period must have been two or three weeks. (Ibid. vol. ii. p. 674.)

[3] Noah Webster, op. cit. vol. ii. p. 48. To some extent Schnurrer held that there is some connexion between measles and Influenza.

[4] See Biermer, op. cit. p. 619, for references on these points.

So also the very imperfect knowledge we have of the relative prevalence of the acute inflammatory affections, makes it quite uncertain whether cases of simple bronchitis, rheumatism, and pneumonia really lessen in number during Influenza. According to Graves (Clinical Med. vol. i. p. 425), during acute diseases persons are less liable, but they may be attacked at convalescence.

It has not been shown to prevail especially in years when intermittents have been more common, yet there may be some connexion between the diseases (see *Symptoms*). Instances have been given in which intermittents seemed to disappear, and others in which the Influenza seemed to cause intermittents.

Relation to the Diseases of Brutes.—In some epidemics of Influenza, dogs,[1] horses, cats, and, it is said, birds, have been affected simultaneously with an epidemic catarrh. Horses are subject to an epidemic catarrhal disease (1827[2]) even when no Influenza prevails among men, and this disease, to which veterinary surgeons now give the name of Influenza, appears closely to resemble human Influenza. Its contagiousness has been warmly debated, and it certainly appears incapable of inoculation, but yet some believe it to pass from horse to horse. It appears to be now generally thought an epizootic affection, and dependent on a specific cause.[3]

After this statement of the facts connected with the spread of Influenza, we proceed to notice the speculative subject of the

Nature of the Exciting or External Causes.—So enigmatical are the phenomena connected with Influenza, that caution is necessary in attempting to form some idea of what the nature of the exciting cause may be.

It must be a specific agent of some kind. From the earliest times authors have come to this conclusion; the similarity of the symptoms in different epidemics show that this agent is the same in its successive invasions. If it be connected with any unusual meteorological or atmospheric condition, this has not been detected, and cannot be at present even guessed at.

At the same time this agent must be in the air; the diffusion is too rapid to suppose it to be conveyed by water; besides, water-poisoning is usually localized. It cannot be attributable to food. There remains only the air as a medium of communication; and that this is so, seems also shown by the way in which it can attack vessels at some distance at sea.

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[1] In the great epidemic in Australia in 1851-52, dogs were affected in great numbers.

[2] In this Influenza of horses, which spread over almost all Europe, no cause could be found in the weather, food, or work of the horses; transfusion of blood of a diseased horse did not communicate it to another; many veterinary surgeons considered it to be contagious; others did not hold this opinion. Influenza prevailed among men in North America, Mexico, and Siberia, but not in Europe.

[3] Turpentine has been used beneficially in the so-called Influenza of horses.

There is, then, some special agent in the air. But this cannot be a gas; no gas could spread in this way without utter dispersion and destruction. Besides, the manner in which it is located in a part of a town, a street, even one side of a street, for a time, or affects a town without touching a village a mile or two off, is quite conclusive against the hypothesis of seleniuretted hydrogen, allotropic oxygen, or any other gas being the cause. Nor can it be any molecular matter driven through the air, arising from some unknown telluric source, for this would be equally diluted and dispersed. The agent evidently cannot own one single and primary origin; it may, indeed, issue from one spot, but all the phenomena of its spread show that it must, in its transit, reproduce itself. Otherwise, if a gas, it must be rendered innocuous by dilution; if an organic matter, by oxidation; if a suspended mineral matter, by subsidence. It must increase, and the more the subject is gone into, the more firmly will the idea gain upon the mind, that there must be a continual reproduction of the agent, to a greater or less extent, in different places.

Now this reproduction must either be in the air or in the bodies of the sick, in which latter case the agent would be a true contagion. If it grows in the air, the only conceptions we can form are, either that some force changes successively the atmospheric elements in some way, or that the increase is a vital one, and consists of microscopic plants or animals. The first idea is supported by no evidence; and as to the second, we find ourselves in the presence of the so-called fungoid theory of Influenza. There are many phenomena consistent with the hypothesis of a vital and growing cause: the occasional introduction of the disease by persons about whose bodies or clothes the fungi may cling; its passage at times with the wind, contrasted with the occasional passage against it when other modes of conveyance may be presumed to come into play; the gradual development of the disease to a climax, and then its decline, contrasted with its occasional persistence when the conditions of growth may be supposed to be more persistently in the same place; the entire disappearance of the disease, and its extremely rapid resuscitation when it again appears; its birth, apparently in various parts of the world, and yet its evident incapability of originating in some countries, as France and England (whither it has always passed from other lands), are all easily explicable if we assume a fungoid origin, and remember the different conditions which can effect the development of fungi. The remarkable powers which have lately been ascribed (with what justice time must show) to those lower forms of life, increase the interest with which this question must be regarded.

But, on the other hand, there is a complete want of direct evidence, without which the argument in favour of a special living agency is worth little. It is impossible to make a certain and assured step without some tangible evidence.

Moreover, for the rapid increase of fungi we should suppose certain meteorological conditions to be necessary—a certain temperature, moisture, organic effluvia; but the spread of Influenza has little, if any, connexion with these conditions.

If the cause be a fungus, or some allied organism, it may increase in the body as well as out of it, and if so would be found in the secretions, especially in the nasal, buccal, and bronchial mucus. In this way human intercourse would spread it. A thorough microscopic examination of these discharges is yet wanting, but possibly the next epidemic may supply this link.

If the agent is not a fungus, the only other ready explanation which presents itself is that of a true contagion; namely, that particles of the sick body being thrown off are in some special condition, or are undergoing certain putrefactive or other chemical changes, which can excite a similar action on particular parts of other human bodies. And in this case, to account for the spread of Influenza, we must believe that these particles pass off in myriads from each sick person, are excessively small and light, perhaps become dried up, and floating through the air, to greater or less distances, are breathed or swallowed by other persons, and then set up in their bodies the same series of changes which the particles themselves are undergoing. This view seems to me to involve greater difficulties than the fungoid theory, *i.e.* it accounts less satisfactorily for the spread of Influenza.

If neither of these views be correct, then the cause of Influenza is something of which we have no conception whatever. It seems to me to be impossible at present to come to any conclusion as to the nature of the cause.

Predisposing or Internal Causes.—Race has no influence, sex probably none, or, if at all, women are slightly more affected; age has only a slight effect; young children are, it is said, rather less affected than old persons. If any special bodily predisposition is necessary, it is common to the whole human race, and apparently to horses, dogs, cats, &c. herein differing greatly from several of the true contagions.

Persons in overcrowded habitations have, particularly in some epidemics, especially suffered, and several instances are on record of a large school or a barrack for soldiers being first attacked, and of the disease prevailing there for some days before it began to prevail in the town around. Sometimes, on the other hand, schools and prisons have escaped.

A low, damp, ill-ventilated and unhealthy situation appears to predispose to it,[1] and in some instances, in hospital patients, it has assumed a malignant character (Sir George Baker, Gray). In other cases again, hospital patients have escaped; for example, the old people in the Salpétrière in 1837, when the younger attendants were attacked.

It has been supposed that persons with chronic lung diseases, especially emphysema, and chronic heart affections, are particularly liable, but this seems uncertain; it is probable that the Influenza being more serious in such persons, creates the impression that they are as a class more liable. The Registrar-General has shown that in 1847, the increase of deaths by Influenza was much greater in the districts in which ordinarily there is a high mortality than in healthier places; this must indicate either greater prevalence or greater severity of the disease.

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[1] Pearson noticed this in both 1762 and 1782.

SYMPTOMS.—*General Course of the Disease.*—The symptoms of Influenza are compounded of two conditions—a general fever of determinate duration, and a marked and evidently specific affection of the mucous membrane of the nose, mouth, throat, and respiratory tract, which has also a determinate course.

Individual cases differ in the proportion of these two conditions, and in addition there may be superventions of true inflammation of the lungs or pleura, or implication of other mucous membranes, those of the stomach and intestines in particular, and less frequently of the bladder and kidneys.

It would appear that the fever has the priority, and that shivering or coldness down the spine, with heat and flushing and dry skin, quick pulse, thirst, and severe headache, very frequently usher in the attack. These symptoms precede any local signs. But it would be very desirable to re-investigate this point. The febrile symptoms sometimes come on quite suddenly, sometimes develop slowly, in from twelve to thirty-six hours, or even to four days. When they commence suddenly, the first symptom is often an extreme frontal headache, with pain and aching in the eyes.

They last for four or five days usually, or sometimes a few days longer, and then disappear gradually, or occasionally rather rapidly, with profuse perspirations, or spontaneous diarrhœa. Sometimes they continue ten or twelve days, but this is generally when pneumonic complication supervenes.

The specific catarrhal affection usually follows the early symptoms of fever; sometimes occurs at the same time, perhaps sometimes precedes them. It appears to commence in extreme hyperæmic swelling and dryness of the mucous membrane of the frontal sinuses, the nose, and, in a less degree, of the conjunctivæ, causing intense pain across the brows, great sneezing, sometimes epistaxis and thin acrid discharges from the nose and eyes; the same condition then occurs in the pharyngeal, the laryngeal, tracheal, and pulmonary mucous membrane, to the minutest ramifications. Usually, perhaps, the affection commences above and passes rapidly down, but sometimes the whole tract is attacked at once. The inside of the mouth and the tongue are also, but less, affected, and the pharynx is also not so marked by hyperæmia as the other parts. A punctiform redness of the mucous membrane of the palate, something like the eruption of measles, has been lately described by Tigri, and considered to be pathognomonic. The discharge from these membranes, when it occurs, is first thin and acrid, and sometimes bloody; it becomes afterwards thicker, more tenacious, and at length purulent; great sneezing, sore throat, difficulty in smelling, violent paroxysmal cough, pains in the chest; occasionally very sharp stitches in the side, which are apparently often nervous, and not pleuritic, accompany the specific condition of the respiratory tracts. Great dyspnœa and the stethoscopic examination show that there is immense congestion of the lungs, and often the face and lips show very considerable impairment in the aëration of the blood. In pure cases the catarrh is at its height on the second and third, or fourth day, and declines about the fifth to the seventh; but cough expectorations often remain for some time. In severe cases the disease lasts with great severity even to the tenth or twelfth day.

Attendant upon these symptoms, and in proportion, it is usually supposed, to the fever, though some have thought it to be in more direct ratio to the extent and violence of the membranous catarrh, is a peculiar state of the nervous system. Very early in the disease there is a remarkable nervous depression, loss of strength, and lowness of spirits, combined often with great aching in the muscles, and severe nerve pains in different parts, which certainly give one the impression that both muscles and nerves are undergoing some profound nutritional alteration. The mind, too, becomes weak, and sometimes there is even stupor or delirium. In some epidemics, indeed, the early sopor or cerebral heaviness is very remarkable.

These nervous symptoms often last longer than either the fever or catarrh; hence convalescence is tedious, and mental activity slowly regained.

In pure cases, when the disease is over, the nasal and respiratory mucous membranes do not for some short time entirely recover their structure, at least if it be true that there is increased liability to common catarrh. Also if it be true that there is a greater liability in future epidemics of Influenza, it is possible that some structural change may permanently remain. The severity of the cases differs greatly, and sometimes the affection is very slight, sometimes very severe.

Consideration of the Special Symptoms.—1. *Temperature of the Body.*—No observations have yet been made with the thermometer. In some epidemics (1580, Salius Diversus) there has been intense heat of skin; in others (1775, Fothergill) the skin has not been particularly hot. But perhaps this might depend upon individual cases; for in the same epidemic some have great, others have slight, fever.

2. *Condition of the Skin.*—Sweating at first is usually absent or partial. If it is profuse in the early stages, the disease is sometimes arrested. The perspiration is often sour smelling, and is said to be very acid. In the epidemic of 1782 in London, the sweating was so profuse as to cause the name of sweating sickness to be given to the Influenza. Sudamina are sometimes seen in great numbers, so that the case looks like miliaria. A pustular or herpetic (Peacock) eruption about the mouth sometimes occurs. There is no decided

eruption peculiar to Influenza, but occasionally it is said rose-coloured little blotches, and sometimes urticaria, are seen. There is sometimes most decided hyperæsthesia of the skin of the neck and head; this is usually coincident with severe headache.

3. *Nervous and Muscular Symptoms.*—The headache is often excruciating; frontal most usually; limited to the region of the frontal sinuses, or extending more or less over the head, or over the face (Antrim of Highmore); there is often great heaviness, sometimes torpor, and occasionally delirium. In some epidemics high delirium has been considered a mortal symptom (Huxham in 1737). Severe vertigo is a common symptom. There is a general lowering in the acuteness of all the special senses. The spirits are low, mind weak; the nights restless, and this loss of sleep is not in relation to the fever; it is seen often in patients without fever.[1]

Meningitis occasionally occurs, and sometimes otitis, and there is often severe pain in the region of the Eustachian tube. There are also neuralgic or rheumatic-like pains of many parts of the body, especially of the muscles of the neck, loins, legs, and the intercostals.

The extreme prostration of muscular strength has been already noticed; it is often a very early symptom, and in some epidemics has given almost a special character to the disease; the complete return of strength does not occur till after convalescence is far advanced.

4. *Respiratory System.*—The paroxysmal cough is one of the most distressing symptoms, and sometimes causes hernia, or abortion in pregnant women. At first dry, the cough is soon attended with stringy, often bloody sputa; as soon as the sputa get more consistent, thicker, more opaque, and purulent, the cough lessens. In different epidemics the amount of cough has varied, but this, may be, in part depends on erroneous observations, as formerly no doubt epidemic hooping-cough was confounded with Influenza. Dyspnœa is often considerable, and is dependent either on the great congestion of the respiratory tract, or on pneumonic complications, or possibly, as suggested by Graves, on some special implication (paralysis?) of the vagus. There are often remissions in the dyspnœa not accounted for by stethoscopic signs. Occasionally there are orthopnœa and suffocative attacks. Sometimes there is intense and oppressive feeling across the chest. The number of respirations is often great, and the pulse-respiration ratio becomes one to two and a half or one to three. In bad lung cases the voice is often very weak as well as hoarse.

At first the stethoscopic signs are almost wanting; the vesicular murmur is feeble, even though the percussion note be clear; if there be dulness, it is equal and indetectible. Afterwards when œdema of the lung occurs there are fine moist râles, and sonorous and sibilant rhonchi are present in some cases.

Capillary bronchitis, pneumonia (which is usually combined with pleurisy), and pleurisy are present in some cases, though it is impossible to state in how many. It has been supposed that pneumonia occurs in from 5 to 10 per cent.[1] In some epidemics pneumonic complication is supposed to be more common, as in 1837; the pneumonia is said to be of the catarrhal variety when it occurs during the attack (about the fourth to the sixth day), and of the croupous kind when it occurs, as it sometimes does, in convalescence (Lombard). The supervention of pneumonia is not easily detected by stethoscopic signs before consolidation, in consequence of the œdema. The same reason makes it sometimes difficult to detect true capillary bronchitis unless one lung is more affected than the other. Pleurisy is easily detected.

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[1] The epidemic of 1712 was attended in Tubingen by great drowsiness, and in that outbreak the brain symptoms appear to have been unusually heavy.

Sometimes it is supposed that a sort of paralysis of the lungs occurs with great œdema (Graves), possibly from affection of the vagus. Collapse of some portion of the lungs often occurs. That during the height of the disease aëration is most imperfect, is evident from the dark lips, congested cheeks, and great distress, which are often seen.

As sequelæ to the chest affection, chronic laryngitis, chronic bronchitis, emphysema, and tuberculosis are sometimes seen. Yet it is well known that some phthisical patients pass well through Influenza without increase of their disease.

5. *Circulatory System.*—At first strong and quick, the pulse soon becomes soft, and in the latter stages feeble, and even slow. It is often singularly changeable within a few hours. Heart affections are not common, yet pericarditis will occur, and is then usually complicated with pleurisy.

The blood is buffed and cupped in pneumonic complications, perhaps in all cases. (Vigla.)

6. *Digestive System.*—Nausea and vomiting are sometimes seen in the commencement; diarrhœa is much less frequent till towards the end, when there are often rather profuse discharges; thirst and complete anorexia are very usual. There is sometimes pain in the right hypochondrium, and a yellowish tint of eye and skin. (Peacock.) In some cases there is a decided icteric state of the skin. The great depression and languor is very similar to that which accompanies some cases of jaundice when the bile is accumulating rapidly in the blood. Sometimes the bilious vomiting, fever, and oppression of the brain cause the case to resemble the bilious fever described by authors. There is no evidence of any splenic affection. In some epidemics these gastro-enteric symptoms have been, it is said, more pronounced than in others, but there is no doubt that cases of typhoid fever complicated with or following Influenza have often been described. As a rule, in pure cases, the symptoms of stomach and bowel implications are not marked, or are caused by medicine or food.

Urinary System.—The urine is at first scanty and high coloured; at a later period it becomes sedimentous from lithates, which are often pink; it is believed there is no albumen nor bile, but good observations fail on these points, as well as on the composition of the urine in twenty-four hours. Occasionally there is almost complete or entire ischuria.

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[1] Biermer, op. cit. p. 624.

Genital System.—The catamenia are sometimes induced, and amenorrhœa has been thus cured. Abortions are frequent, especially in some epidemics, probably from the violence of the cough.

Lymphatic System.—Swellings of the parotid, the submaxillary, and sometimes the cervical glands are observed, and occasionally, but rarely, severe parotitis follows.

It is somewhat curious that either an intermittent fever has been united to Influenza in some epidemics, or that the Influenza has had an intermittent character. Thus in 1580 Sennert mentions that the quartan fever was joined to the epidemic; in 1658 Willis states that the epidemical catarrhal fever often had an intermitting character, usually tertian, rarely quotidian. In 1762 Baker says that Influenza appeared under the form of an intermittent with tertian periods. In 1767 Donald Monro also saw an intermittent character, but not so marked as in the epidemic at Bremen in 1762. In 1775 he says that few persons had such distinct paroxysms as to resemble those of an ague; but Fothergill (1775) states that "in many instances the disease assumed the type of an intermittent towards its decline." In 1803 Pearson noticed that the lassitude and depression which continued after the fever had gone had an intermittent character, and were worse every other day. The histories of the recent epidemics show no character of this kind, and it is possible that in former centuries the far greater prevalence of malaria impressed on other diseases a periodical character which was not in their own nature. But the observations are curious in connexion with the opinions of those who have connected Influenza with malaria.

VARIETIES OF INFLUENZA.—The varieties in different epidemics have been already referred to. In the same epidemic Influenza differs in intensity in different people. In some persons it is an extremely slight disease; in others, a very severe one; this is especially the case if there are pulmonic or gastric complications. So also in some cases an unusual nervous depression prolongs a case which might be otherwise a mild one, or paroxysmal cough and expectoration, or flying neuralgia, or rheumatic-like pains continue for some time during convalescence.

MORTALITY.—This seems to vary greatly in different epidemics (1837 and 1847 were more fatal than 1833–4; Graves), and is also partly, perhaps, dependent on treatment. Wierus says that the great mortality in Italy in 1580 was owing to the promptitude with which the Italians bled; the mortality in London has sometimes been severe, while it has been slight in Germany. In 1837 the rate of mortality was calculated at two per cent., but it was considered that this was a very severe epidemic.

There appears no doubt that mortality increases greatly with age. It is also higher in persons with chronic bronchitis, emphysema, and chronic heart-diseases, especially dilated and fatty hearts. Mere valvular disease, without loss of power, has little influence. During the prevalence of Influenza, other causes of death show an increase, especially pulmonary complaints, and typhoid, and typhus. This depends probably on the supervention of Influenza upon those affections.

DIAGNOSIS.—If the term Influenza is restricted to the truly epidemic disease which spreads over large tracts of country, there is no difficulty in the diagnosis. Although there is no special eruption as in the exanthemata, or peculiar cough as in pertussis, or membranous pellicle as in diphtheria, the collection of symptoms is peculiar.

Nor can there be any confusion between cases of epidemic cough and isolated cases of catarrh, arising usually from marked meteorological conditions. However common such attacks, however severe and *influenzoid* they may be in certain cases, they do not constitute an epidemic; there is no disease spreading over the country. Moreover, the symptoms are really dissimilar in their mode of connexion and succession. Far more difficult is the diagnosis between true Influenza and catarrhal fevers invading a town or district. That there are such local or endemic attacks of catarrhal fever seems certain, and it is doubtful whether or not they should be classed with Influenza. They want the power of travelling; they attack more slowly, and are far less common among the population. There is for the most part less of the overwhelming prostration, and fewer mucous membranes are attacked. They appear usually to be merely the common catarrh developed into unusual proportions by changeable or severe weather, and possibly this may be the simple explanation of their occurrence. If by the use of the thermometer a typical course of temperature is discovered in Influenza, or if the examination of the excretions detects any special characters, the diagnosis will be easy. Till these points are determined some doubt must exist, nor does it seem to me possible to lay down any precise rules of diagnosis. There are few points more deserving careful study than the precise characters and causes of catarrhal fevers, localized in a town or district, and not forming part of a general epidemic.

There is no other disease with which Influenza can be confounded, but during its prevalence many other diseases—bronchitis, typhoid fever, &c.—are often called Influenza, and this probably has given rise to the opinion that during epidemics of Influenza such diseases lessen, to reappear at its close.

PATHOLOGY.—We are not yet in a position to discuss the pathology of this disease. Does the agent enter the blood, act on the nervous system, and then by election seize upon and irritate the mucous membrane of the respiratory tract? Or is it really a membranous local disease, acting very promptly (just as simple angina will act on the system at large) in the secondary constitutional effects? At present the sequence of symptoms seems to show the first view to be more probable: viz., that it is a general disease, with a special secondary localization.

What is the exact nature of the general disease? The blood is buffed and cupped; *i.e.* there is increase in the fibrine; that is all that is known of the blood; the peculiar changes in the nervous system and the muscles are quite unknown.

What is the exact nature of the respiratory affection? If we reply, it is a general hyperæmia, this is a mere translation of terms. Of the exact cause of that hyperæmia we have no idea. Is there a partial coagulation of blood in the venous system, or some affection of the vaso-motor nerves leading to general dilatation; and is the altered mucous discharge due to such change, or to some special condition of the nutrient plasma as it comes from the vessels, which strikes deeply at their growth and nutrition? The inflammation, if we are to give it that term, is evidently specific; in what the specific character consists it seems at present vain to inquire.

MORBID ANATOMY.—Fatal cases of pure Influenza are rare; they occur chiefly in old persons, with old lung or heart disease, or in consequence of recent inflammatory pulmonic or cardiac complications.

The results of simple Influenza seem to be general congestion of the respiratory tract, amounting sometimes to enormous congestion of the lungs, œdema of the lungs, with more or less collapse. The collapsed portion is smooth, non-crepitating, and is said to be sometimes softer than usual, like gangrene, but without fœtor.[1] Sometimes membranous exudations are found in the bronchi, not unlike those of croup.

If pleurisy and pneumonia have occurred, the usual post-mortem appearances of those diseases are present. The pneumonia is sometimes lobular (or possibly this statement has arisen from lobular collapse not being identified) or lobar, and is often double.

PROGNOSIS.—The very young and the very old bear Influenza badly, especially the latter. Persons with chronic bronchitis, emphysema, and fatty heart, are bad subjects.

In persons without such complaints the danger is chiefly connected with the state of the lungs. Great dyspnœa, very weak voice, impossibility of coughing up the tough sputa, and duskiness of the face, are unfavourable signs. If the pulse becomes early very feeble and slow, and then unequal and intermittent, it shows that the heart is not receiving its due supply of blood on account of the lung-congestion.

The fever and the nervous symptoms seldom kill, yet in some epidemics there have been frequent delirium, convulsions, and fainting, and these have always been found to be very bad symptoms.

As favourable signs may be noted, copious warm sweats, loose so-called concocted sputa, spontaneous diarrhœa, and urine with copious red lithates.

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[1] Especially in very aged persons: Greene, in Graves' Clin. Med. vol. i. p. 438.

In the case of pregnant women there is danger of abortion and subsequent hæmorrhage.

TREATMENT.—*Preventive Treatment.*—No means are yet known by which Influenza can be prevented. Unfavourable hygienic conditions, and especially overcrowding, heighten its prevalence and its severity; but persons in the most favourable circumstances may be attacked. Perhaps persons in well-warmed and yet ventilated houses escape best. It has sometimes been noticed that persons exposed by work to the weather suffer most: hence it may be a rule that those persons who can do so, should be more within the house during an epidemic; but as bed-ridden persons are not infrequently attacked, this is no guarantee.

Treatment of the declared Disease.—*Regimen.*—It is of great importance to have the room cool and well ventilated. Pearson, whose little work on Influenza is one of the most practical which has ever been written, pointed out, in 1803, the difference in this respect between common catarrh and Influenza. In the former case the patient is better in bed and in a warm room; in the latter case, if the patient is not too ill, it is better to get him out of bed after the third day, and to place him on a sofa.[1] Draughts or chills must be, however, most carefully avoided, on account of the risk of pneumonia.

As there is usually almost complete anorexia it is difficult to give much food. The common custom of giving hot beef-tea is an extremely bad one; it invariably increases the headache and languor, and, as Pearson pointed out, any warm food which forces sweating appears not only to be useless, but to do harm. Solid meat also should be abstained from for two or three days in bad cases. Several writers recommend vegetable food for four or five days. Plenty of cold drinks, especially sub-acid fruits, oranges, lemon-juice, cream-of-tartar water, raspberry vinegar, weak citrate of potash, and citric acid flavoured with sugar, barley-water with lemon-juice, infusion of mallows or althea, and drinks of the like kind, should be given *ad libitum,* and when there is fever they should be iced. Very weak cold white-wine whey is a very grateful drink. Some good writers speak strongly against the practice of stimulants early in the disease, in all young persons; the great languor and weakness often leads to their use, but it seems probable that they do harm. If stimulants seem indispensable, claret or hock, with seltzer water, are the best. In old persons it may be necessary to use stimulants earlier and more freely.

Stimulants must however be given, and often given largely, in the later stages, if the heart fails, and especially if there are symptoms of intense lung congestion and asphyxia. Brandy, with ammonia, must then be freely used.

As soon as the severity of the fever is passing away, patients should be made to eat; the appetite is still bad, but they will generally take food. Care should be taken not to derange the stomach by too great quantity or variety of food, of which there is some danger.

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[1] In some of the older epidemics the practice in England was to keep the patient extremely hot in bed, and to give calefacients. 1801.

No experiments have yet been made, to my knowledge, on the effects of cold affusion in the stage of fever ; but possibly it might be useful. The wet sheet has been used, and apparently with some benefit. For old people, Schönbein used to order warm baths or warm fomentations.

Keeping the air of the room moist, by conducting the steam from a boiling kettle into it by means of a tube, or by putting boiling water into flat shallow vessels, appears to ease the cough. Also, as in common catarrh and bronchitis, the inhalation several times daily of hot steam is most useful. The old inhaler of Mudge, with the hollow handle, and the valve in the cover, or any of the new inhalers, may be used. If they cannot be obtained, breathing through a sponge dipped in hot water is the best way.

Drugs.—Slight cases require almost nothing; a little cooling saline medicine, citrate and acetate of potash, nitrate of potash, &c. .

In severer cases treatment must be more active.

Blood-letting seems always hurtful, and this was noticed so long ago as in the epidemics of 1580. Hardly a writer of any note has failed to make the same remark. The fever is not relieved, the nervous depression is increased, and the risk of the lung congestion and paralysis is augmented. Even with supervening pneumonia, in the old days of bleeding, blood was very seldom taken more than once. If cupping or pneumonia come on with severe pain, a few leeches or a pleurisy glass to the painful part are often useful, but depletion should hardly go beyond this.

A dose of calomel, one to three grains, according to circumstances, repeated once, but not oftener, should be given at first, and may be followed by a saline purgative. Pearson strongly recommended this, and various writers have endorsed the practice. The calomel generally brings away copious dark-coloured motions, after which the patient is much better in spirits, and the fever abates. But neither mercurial nor other purgatives should be too freely or repeatedly given, as the intestinal mucous membrane is irritable. Repeated catharsis is sometimes most injurious. The substitutes for mercury, podophyllin, jalapine, &c., have not yet been tried in Influenza, and it is impossible to say whether they will be more or less useful.

In children, grey powder must be substituted for calomel, or, what seems better for them, clysters of warm water, with a little castor oil, may be used.

From the good effects of one or two doses of mercury some have proposed to continue to give mercury, stopping just short of salivation. But this is bad practice; there is no evidence that these small repeated doses of mercury are useful, and it is impossible to be sure that salivation will not come on before we are aware, with all its evils.

Emetics, at the onset, have been very strongly recommended, and in the older epidemics an emetic of antimony and ipecacuanha was invariably given, when the patient was first seen. If there is much nausea an emetic is useful, and perhaps may be so in all cases; but there is one disadvantage, it occasionally produces great and permanent irritability of the stomach, so that it is afterwards difficult to check the constant vomiting. Tartar emetic has been chiefly used, but it causes much depression. On the whole, it seems undesirable, as a rule, to give emetics.

After the bowels have been well acted upon, the best remedies to give in common cases seem to be nitrate of potash mixed with lemon-juice and sugar. It seems most useful to give it highly diluted, so that it may be taken as drink. From 60 to 120 grains, in twenty-four hours, may be given to an adult.

Supposing the chest symptoms are not urgent, nothing else need be done; but if the lung congestion is considerable and the cough very hard, some expectorants must be used. Of these, ipecacuanha seems on the whole the best, and can be combined with conium or henbane, or with the ætherial tincture of lobelia. (Blakiston.)

Tartar emetic, as an expectorant, has been strongly recommended, but it appears to be too lowering in many cases. In the epidemic of 1847 I found it to be of little service, and sometimes to cause irritation and congestion of the intestinal mucous membrane. It is, I believe, better avoided altogether.

Opium requires to be used in the bad cases with the greatest caution. There has been much discrepant evidence as to its employment, but on the whole it seems, as Pearson pointed out, best to defer its use till the later stages. If given early, it increases the tightness across the chest and the difficulty of breathing. At a late stage, when the expectoration is coughed up easily, and all danger of great lung congestion seems passing off, opium with ipecacuanha quiets the paroxysms of cough, and gives great ease.

Sometimes, however, when the cough is extremely violent, and conium and henbane do no good, opium must be given. In fact the cough itself, simply as a mechanical agent, excites an unfavourable effect on the congested lung, and must be stopped. Then Dover's powder, with nitre and lobelia, should be given; if this does not answer, the liquor morphiæ muriatis or the bimeconate of morphia with ipecacuanha, in large doses, must be used.

Squills seem decidedly hurtful till quite the latter stages.

If there is great tightness across the chest, sinapisms and warm bran poultices or warm water fomentations must be constantly used. Sharp stitches in the side, if pleuritic, must be treated with sinapisms and warm poultices, or, if very severe, with a few leeches, followed assiduously by warm fomentations. If no friction-sound can be detected, they are intercostal neuralgic pains, and are soon relieved by warmth, opium, and chloroform, applied externally.

In the latter stages, if the expectoration is profuse, the cough still violent, and the strength failing, senega and serpentaria, mixed with light wines, seem to be very useful. Ammonia must also be used.

If the expectoration continue extremely profuse, the acetate of lead, with a little opium, is useful.

Some of the older writers thought that cinchona bark was hurtful in the earlier stages, but in some of the late epidemics quinine appears to have been found useful throughout. Whether this be the case or not, it seems clear that immediately the acute stage is passing off quinine should be freely given. It does good service against the neuralgic pains which are often troublesome at the commencement of improvement.

Warm plasters between the shoulders have been much praised by some writers (Legendre). Blisters do no good, and add to the patient's sufferings.

Inhalations have been tried both for the cough, sore throat, and nasal soreness. Pearson used the vapour of ether, which he had found very useful in common catarrh; it was not so good in Influenza. Chloroform, in small quantities, may relieve the tightness and the violence of the cough. Inhalation of steam has been already noticed.

In future epidemics it would seem very desirable to try various inhalations to act on the membranes of the nose, pharynx, and lungs. It is impossible *à priori* to say whether they could be of any use, but small quantities of chlorine, iodine, carburetted hydrogen, even perhaps sulphurous acid, might be tried. The naso-bronchial mucous membrane is very accessible to such influences, much more so than to medicines introduced into the blood.

It may be a question also whether some local applications could not be made to the membranes of the nose and throat, such as solutions of iron, catechu, or alterative substances of that kind. Possibly the local disease might be thus partly checked.

The use of the sulphites of potash and soda may also be suggested as a local application to the throat and nose.

Complications.—It is very doubtful whether pneumonia is benefited by bleeding; the pneumonia has itself a course, and cannot be cut short; it is probably better to persevere with ipecacuanha and nitre, and to apply only a few leeches or a cupping-glass if pleuritic pain be intense.

In double capillary bronchitis bleeding is hurtful; the great danger is suffocation; brandy and ammonia, with valerian and lobelia inflata, must be freely used. Sometimes, even in cases of exhaustion, it is necessary to give an emetic, as the thick secretion blocks up the tubes; sulphate of zinc and ipecacuanha is then the best emetic.

In obstinate vomiting, hydrocyanic acid, and very small doses of morphia, with effervescing draughts, will generally suffice.

Excessive diarrhœa must be checked, but moderate diarrhœa does good, and is indeed a favourable sign, especially on the third or fourth day.

In suppression of urine, a very hot bath and copious draughts of linseed-tea, with a little liquor potassæ, or chlorate of potash, must be given.

If there be intense headache and stupor, purgatives, cold applications to the head and a few leeches, either to the temples or the Schneiderian membrane, will often give relief.

If there be much coryza and great pain in the nose and frontal sinuses, a few drops of solution of muriate of morphia in a little water, sniffed or injected up the nostrils, will give relief.

If rheumatic symptoms come on, colchicum is said to be useful (Peacock) in small doses (4 or 5 minims of the tincture of the seed), given every 3, 4, or 6 hours, with ammonia and opium. Iodide of potassium with colchicum is also sometimes useful.

Convalescence.—Iron and quinine must be given for some time in small doses.

A very nutritious diet, beer, and wine, must be employed. Milk in large quantities is very useful. Milk and seltzer water is a favourite German remedy.

The skin must be very warmly clothed, as it is very sensitive.

If there is much dyspnœa left behind, the alcoholic or ætherial tincture of lobelia should be used. Flying pains of the chest are best treated by opiate fomentations, or a liniment of acetic acid and oil of turpentine, recommended by Dr. Stokes.

If a paroxysmal cough is left behind, with copious and rather viscid expectoration, ammoniacum and opium should be given.

HOOPING-COUGH.

By Edward Smith, M.D. F.R.S.

Definition.—A convulsive cough consisting of a series of forcible expirations, followed by a deep, loud, sonorous inspiration, and repeated more or less frequently during each paroxysm; occurring usually in childhood, and once only during life, and continuing several weeks. Cullen's definition is, "Morbus contagiosus, tussis convulsiva, strangulans, cum inoperatione sonora, iterata, sæpe vomitus."

It is popularly known in England as Whooping-cough, Kink-cough, Chin-cough; in France, Coqueluche, and in Germany, Keuch-husten and Kik-husten; from the sonorous inspiration which marks it; and technically, as Tussis convulsiva (Willis and Sauvages) and Pertussis (Sydenham and Cullen).

History.—It is difficult to believe that a disease having characters so well and easily defined could have been known to the ancients without a description having been recorded by which we might now recognise it; and, as no writer before the middle of the seventeenth century has described it, we are led to the conclusion that the disease was unknown to the fathers of medicine, or that it has acquired one of its chief characteristics since their day. Diseases having a contagious or epidemic character, and resembling Hooping-cough in its catarrhal symptoms, were clearly known to Hippocrates and others before the Christian era, and have been described by Arabian, Italian, and French authorities down to the sixteenth century; but, lacking the distinctive character of the Hoop, they more nearly resembled influenza than any other disease now known to us. Hence the history of the disease cannot be clearly traced back to a period earlier than that of Willis, from whom we have received not only the first description of it, but one which in all respects is applicable to the disease as it exists at this day; yet, as from his definition, "Tussis puerorum convulsiva seu suffocativa, et nostro idiomate, *chincough* vulgo dicta," it is probable that the disease was then commonly known to the people, we may infer that it had existed in England some time before he described it. Dr. Gibb avers, but without citing authorities, that it has been known traditionally among the French Canadians for more than three centuries; and, as they are presumed to have received it from France, he affirms that the disease to which Mezeray gave the name *Coqueluche* in the fifteenth century, was truly Hooping-cough, notwithstanding the absence of the distinctive Hoop in that author's description of it.

Causes.—There is no known specific cause to which it can be attributed; but that atmospheric influences are its chief exciting causes may be inferred from the facts that it has often occurred as an epidemic, and is most prevalent at certain seasons of the year. The imperfection of our knowledge in reference to atmospheric influences, other than temperature, and the absence of registration of the prevalence of diseases which do not end fatally, prevent a more minute inquiry into this relation. The fact that Hooping-cough is, without reasonable doubt, a contagious disease, implies that a *materies morbi* generated, or at least acting, within the body, is communicated from one to another person, and that the atmosphere is also the vehicle for its transmission; but as we know nothing of the nature of this *materies morbi* within the body, so are we equally ignorant of its characters when existing without it. Further, we do not know with any precision the period of incubation during which the communicated poison is imperceptibly acting within the body, but it probably does not exceed ten days.

The influence of childhood in the causation of the disease must also be cited. In our analysis of the deaths from Hooping-cough, published in the Medico-Chirurgical Transactions for 1854, it was shown that Hooping-cough was the most mortal of all diseases of children under æt. 1 year; that 68 per cent. of all the deaths from Hooping-cough occurred under æt. 2 years; and that only 6 per cent. of the deaths were recorded after æt. 5 years.

But here again our knowledge is most limited and vague when we attempt to analyse the conditions attending early life which may be presumed to lead to the occurrence of the disease. It is summed up in the phrase "great excitability or impressionability of childhood," by which all influences are asserted to exert special power at that period. But it applies with equal force to the occurrence of other diseases in childhood which have but little affinity with Hooping-cough in its leading characteristics. Yet it accords well with the generally adopted views as to the immediate cause of the cough, to which we shall presently refer, and is further supported by the fact proved in the paper just quoted, that the disease when fatal prevailed more in females than in males—in the sex in which this special character of childhood is the most marked.

Nature and Seat.—The intimate cause, or the nature and seat, of Hooping-cough is variously regarded, as one of the two leading characters of the disease—the catarrhal or the convulsive—is the more urgent; but with literature rich in authorities the preponderance of opinion is in favour of the essentially nervous nature of the disease. This opinion has been held by Hoffmann, Hufeland, Lobenstein, Löbel, Paldame. Wendt, Jahn, Cullen, Leroy, Guibert, Webster, Pinel, Todd, Gibb, and Copland. The immediate seat of this nervous irritation has been very variously ascribed to the stomach (Chambon and Broussais); to the lungs (Wendt and Paldame); to the diaphragm (Millot); to the pneumogastric nerves (Hufeland and Hoffmann); to the phrenic nerve (Jahn); to the medulla oblongata (Copland); to the brain and its membranes (Webster); and to the general nervous system (Guibert); but several of these authorities included more than one seat in their description.

The most characteristic views of recent date are perhaps those of Guibert and Copland, to be found in the renowned Dictionary of Medicine of the latter. Guibert "considers that a common cough may pass into this affection by having the spasmodic state of the muscles of the larynx and of the diaphragm superadded to it; and, therefore, that spasm superadded to cough constitutes the disease—the state of spasm resulting from the high nervous susceptibility and particular disposition to it existing in children, and from individual idiosyncrasy." "The increased secretion of mucus he refers to an excited state of the mucous membrane of the air-passages . . . existing independently of any inflammatory action . . . the nervous symptoms being the result of the spasm, which he considers the chief agent of the morbid phenomena." Dr. Copland writes: "I believe that the disease is chiefly nervous in the simple cases; that it preserves this character more or less throughout, even when inflammatory complications ensue; and that in the uncomplicated state the nervous affection never proceeds beyond irritation. . . . The inflammatory appearances in the medulla oblongata and base of the brain may be owing to the functional relation of these parts to the respiratory order of nerves which receive the first impression of disease."

The writers of high repute who give greater prominence to the catarrhal or even inflammatory nature than to the nervous character of the disease, are Laennec, Dewees, Guersant, Watt, and Badham. Dawson believed that the inflammation was restricted to the glottis, whilst Desruelles, with many others, regarded Hooping-cough as beginning with bronchial inflammation and advancing to cerebral irritation. Many writers, with Guersant, believe that the inflammation is of a specific kind; but the chief distinction which they draw between this and ordinary bronchitis is the marked character of the spasm, and the other evidences of nervous irritation—evidences which coincide more with the views of those who believe in the nervous nature of the disease, than with those who consider the disease to be essentially inflammatory.

In a disease in which these two main characteristics exist there are doubtless grounds for difference of opinion as to their relative importance, and particularly when their respective influences vary in different cases and in different epidemics, and when observers, by their special studies, are led to regard cases from different aspects, as the nervous, inflammatory, and pathological. Those who adopt the opinion that Hooping-cough is essentially a disease of the blood, and is due to a morbid poison existing in that fluid, regard both the nervous and the catarrhal evidences as of equal importance, but with this difference in their aim—that the former are direct evidence of the action of the poison, whilst the latter are the throes of the system to rid itself of the poison by secretion from the mucous membrane. Without denying the existence of a specific poison, and without admitting that the supposed poison is eliminated by the mucous membrane of the bronchi, we do not doubt that that feature which gives character and importance to the disease is the nervous or spasmodic one, and that in any uncomplicated case, when this has been abated, the disease is shorn of its specific characters and dangers.

SYMPTOMS.—On proceeding to state the symptoms of the disease, it becomes necessary to divide them into two classes: those of the simple and those of the complicated form of the disease.

Simple Hooping-cough.—The early evidences are those of simple catarrh without any, or with scarcely any, febrile complication. They are coryza, secretion from the nose, cough, more or less severe, but not at this stage spasmodic, with frothy and watery secretion from the bronchi, lassitude, restlessness, and some diminution of appetite. After a period varying from one to two weeks the cough becomes a more marked symptom,—is louder and more prolonged than an ordinary cough, and generally assumes a spasmodic character. When the nature of the disease has become quite clear, the cough is found to occur in paroxysms, during which the body is bent forward, and a series of short, very rapid, and violent expirations occur, and are continued until the face is extremely suffused and the respiration seems almost to have ceased, when a deep, prolonged, loud, and crowing inspiration takes place. This alternation occurs two, three, or more times in each paroxysm. The attack terminates with the emission of a somewhat large quantity of semi-transparent glairy and very tenacious mucus, which hangs about the mouth and lips, and not unfrequently with vomiting. At a yet later period the pertinacity of the expiratory effort is diminished and inspiration occurs more frequently, whilst the secretion, although still abundant, is more opaque and less tenacious, and vomiting less rarely occurs.

During this period the peculiar character of the sounds with the cough somewhat subsides, and in progress of time it is omitted from some of the attacks—the relative frequency gradually diminishing until it altogether disappears, and the cough has no longer any special characters. In mild cases the disease may soon end; but in more severe cases there remains much exhaustion and emaciation, with defective appetite and increased sensibility of the stomach, which leads to vomiting from trivial causes. The rate of pulsation is increased in a most marked manner during the attack, so that in very severe paroxysms it is too great to be counted; yet it is not due to any inflammatory or febrile condition, but to the mechanical interference with respiration. In the intervals it assumes a normal state, except when the system has become much enfeebled. The force of the heart's action is the greatest at the commencement of each paroxysm, and diminishes sensibly when the rapidity of pulsation is the greatest; and it is also lessened when the disease has been prolonged and the system much exhausted.

The skin is usually soft, and at the end of a paroxysm is bathed in perspiration. It is also usually cool and highly sensitive to low temperature.

Bleeding from the nose is a very frequent attendant upon a severe attack of Hooping-cough; and whilst it shows how great is the interference with the circulation, it is often a most valuable remedy.

The period of the occurrence of a paroxysm is uncertain, but it is particularly liable after a meal, when the stomach is full and the action of the diaphragm is impeded, and when food of slow digestibility has been eaten. If the child be very young, the cough is excited when the nurse throws it about; and if older, crying or seeing another in a paroxysm will bring on an attack. The paroxysms are more frequent in the day than in the night. Hence there is usually much interference with nutrition, and consequent loss of flesh, whilst the lassitude extends to exhaustion and prostration of the system.

In ordinary cases the child regains much of its spirits and healthful appearance between the paroxysms, and runs about, plays, and eats almost as in health; but when the paroxysms are severe, the face remains suffused, the eyes injected, and the surrounding parts swollen during the intervals; whilst loss of strength is proportionate to the constitutional feebleness and the early age of the child, the vomiting, and the duration and violence of the disease.

In a typical case the catarrhal symptoms, without spasmodic cough, continue about two or three weeks, and the spasmodic cough three to four weeks; whilst after the spasm has ceased and the cough has become again catarrhal, the duration may be short, if the child have not been too much enfeebled, and prolonged for some weeks, if otherwise.

Complicated Hooping-cough.—The complications are of two classes, viz.: when Hooping-cough supervenes upon another disease and complicates it; and when, the Hooping-cough being primary, other diseases arise in its course. The former class is a somewhat extensive one, and is for the most part limited to diseases which involve bronchial affections in their course; but the latter only will be considered here.

The complications are of four kinds, viz.: disease of the lungs, disease of the brain, infantile remittent fever, and vital exhaustion. The last may by some be regarded as one of the sequels of uncomplicated Hooping-cough; but when it is considered that the almost infinite proportion of the cases of simple Hooping-cough end favourably, with only a moderate state of exhaustion, it will be thought better to regard the very exceptional occurrence of fatal exhaustion as a complication rather than as a sequel to the simple form. The pulmonary complications are, congestion of the lung, emphysema, atrophy, bronchitis, and broncho-pneumonia.

A certain amount of congestion of the lungs is found in all severe cases of Hooping-cough. It is due perhaps exclusively to interference, through the respiration, with the pulmonary circulation, and it is one of the sources of danger attending the disease; but in the degree in which it becomes a complication, the dyspnœa and frequency of respiration are increased and continue during the intervals, the discoloration and suffusion of the face are more marked, the pulse is feeble and rapid, and the exhaustion of the system is greatly increased. Hæmoptysis of a more or less severe kind sometimes occurs and yields temporary or permanent relief. Physical examination of the chest shows that the respiratory sounds are somewhat more feeble than in simple Hooping-cough, and there may be a shade of dulness on percussion; but unless effusion occurs into the lung-parenchyma, the physical signs are not very marked. There are not any marked signs of fever.

Emphysema, although usually regarded as a sequel of the disease, is a frequent concomitant of the severe forms, and particularly in the children of parents who have been afflicted with chronic bronchitis. Its production is mechanical, as in the case of adults, and occurs from the forcible compression of the air in the lungs, which is effected by the diaphragm and other expiratory muscles, whilst an obstacle is opposed to the egress of the air. This obstacle is most commonly only the ordinary one which exists in the larynx and pharynx, and is a necessary part of the act of coughing, as shown in my paper on the "Closure of the Larynx at its Upper Orifice," in the *Journal de Physiologie*, and as seen at the Rima Glottidis by the laryngoscope; but it may also be produced by the plugging up of a large bronchus after the part of the lung to which it leads has been distended with air. The result of this condition is to increase the dyspnœa and to render it permanent in proportion to its extent, and if it exist in any considerable degree the respiratory sounds will be lessened and the resonance on percussion increased.

Atrophy of a part of the lungs is a not unfrequent complication of Hooping-cough. It results from closure of one or more divisions of the bronchi, by which the ingress of air to a part of the lung is prevented, and the space thus left unoccupied is filled up by the undue expansion of the adjoining cells. When the part thus rendered useless is considerable, the gravity of the complication is great, and it may be detected by the diminished expansion of the intercostal space, and by the absence of respiratory sounds over the part atrophied; but when it is small, the encroachment of the adjoining structure prevents the occurrence of distinct physical signs.

Bronchitis and broncho-pneumonia are, however, the more frequent and fatal lung-complications of this disease. In the paper already quoted from the Medico-Chirurgical Transactions it was shown clearly that deaths from Hooping-cough were almost exclusively due to these diseases, and that they did not correspond at all with the rate of mortality from zymotic or nervous diseases. In both there are evidences of fever in the varying degrees of heat and dryness of the skin, and in the rapid pulse both during the paroxysms and in the intervals. The cough is more frequent and not always spasmodic, and the dyspnœa is more permanent. Discoloration of the face, enlargement of the opening of the alæ nasi, difficulty in speaking, and panting respiration are more perceptible as the complication is severe. The only change in the physical signs is an increase in the moist râles, whilst with broncho-pneumonia there is a more or less persistently localised state of this sign accompanied by some amount of dulness on percussion and lessened respiratory sounds. The general exhaustion and loss of appetite are more apparent.

The brain complications are convulsions and hydrocephalus. It has already been shown that, in the opinion of very able physicians, irritation of the brain and its membranes, and particularly of the medulla oblongata, is so common as to be an integral part of the disease. Usually, however, there are no signs of this state other than the reflex condition which excites the spasm of the glottis; but in no inconsiderable number of complicated cases convulsions occur with or without hydrocephalus.

The occurrence of convulsions cannot usually be predicted; but if the child be teething or suffering from derangement of the bowels, if during the spasm the thumbs be drawn inwards, and during the intervals the discoloration of the face continue without lung-complication, and if there be a marked degree of exhaustion or oppression following the paroxysm, or the eyes be intolerant of light, this complication may be imminent. They are in some cases due to irritation of the membranes of the brain and medulla oblongata, and in others to congestion of the brain, due mainly to interference with the pulmonary circulation.

Hydrocephalus is so frequently a constitutional affection that the relation of Hooping-cough to it is rather that of an excitant of a pre-existent predisposition. The signs are often obscure at first, but in many cases the occurrence of drowsiness, headache and starting during sleep, convulsions, increased heat of skin and rapidity of pulsation, intolerance of light and lessened mobility of the pupils; and in others the persistent disposition to vomiting on being moved, will indicate the advent of this most important complication. The breathing is more irregular and accompanied by sighs, than it is in simple Hooping-cough; and if there be no lung-complication existing at the same time, the diagnosis will not long remain doubtful. When the convulsions are restricted to one side of the body, or when paralysis of one side occurs either with or without simultaneous convulsions on the other, the evidences are still clearer.

The complication with infantile remittent fever is most generally found when the latter disease prevails, and when there have been evidences for some weeks of a disordered state of the bowels. The tongue is coated, the breath foul, the evacuations unhealthy, and the bowels are tender on pressure and tumefied. The patient does not recover health and strength, but, with or without introductory rigors, slowly exhibits signs of fever, having the exacerbations and remissions distinctive of remittent fever, and wanting all the diagnostic signs of bronchitis. Such cases are usually protracted in their recovery, and demand very careful and able supervision.

The complication of excessive exhaustion is most frequent in children of very weak constitutions, or in those which have been enfeebled by previous disease. In such, prostration is a marked feature even during the catarrhal period, but when the spasm has fairly set in it is extreme after every paroxysm. Careful examination into the state of the lungs, brain, and bowels fails to offer any satisfactory reason; and this, with the absence of fever, suffices to indicate the complication to which we refer. Moreover the skin is unusually soft, cool, and liable to perspiration, and the appetite is inadequate to the nourishment of the system.

DIAGNOSIS.—The distinguishing feature which marks Hooping-cough is undoubtedly the paroxysm of spasmodic cough, whether the sound accompanying it amount to a distinct whoop or not; whilst the accidental (as opposed to essential) symptoms are the preliminary catarrh, the glairy tenacious secretion from the bronchi, the early age of the patients, and the general course of the increase and decline in the severity of the paroxysms. In the early catarrhal stage, it cannot be distinguished from a common cold.

PATHOLOGY.—The pathology of this disease has been already hinted at in our account of its history. In reference to simple Hooping-cough the aim has been to determine the cause of the spasm of the glottis and the closure of the larynx, with the prolonged expiratory action of the diaphragm, which are the prominent features of the disease. All writers have regarded the nervous system as the source of this influence, and have speculated upon the part which was principally involved. Thus the recurrent laryngeal nerve as affecting the larynx, the phrenic as controlling the diaphragm, the medulla oblongata as controlling all the respiratory movements by the pneumogastric nerve, and the whole brain and its membranes, have each in their turn been cited as the seat of this disease. The blood has by others been assumed to contain a zymotic poison, which acting upon the nervous system excites spasm, and upon the mucous membrane of the bronchi causes cough, with a secretion whereby it is to be ultimately cast out. We cannot yet, however, arrive at a sound conclusion upon this question, although there are many points of striking similarity between Hooping-cough and some recognised forms of zymotic disease.

The interference with the circulation of the blood which occurs when the respiration is so greatly impeded and the lungs so largely emptied of the residual air, as in severe cases of Hooping-cough, is doubtless the cause of the complications both in the head and the lungs to which we have referred; whilst the exhaustion which follows each paroxysm, and the interference with nutrition, are the causes of the remaining complications. The mechanical act of coughing whilst the larynx and some of the bronchi are closed produces emphysema of the lungs and dilatation of the bronchi; and closure of the tubes by secretion may lead to atrophy.

Morbid Anatomy.—In nearly all fatal cases death occurs not from the Hooping-cough, but from its complications; and the morbid signs will therefore be those of the supervening diseases, and must be sought for in other parts of this work. Dr. Copland believes that in all cases there are inflammatory appearances about the medulla oblongata; and it cannot be denied that congestion of the bronchial, laryngeal, and faucial mucous membrane is always present. From what has been already stated, it may be affirmed that there are evidences of bronchitis or of broncho-pneumonia, with atrophy or collapse of lung in by far the greater number of fatal cases.

Prognosis.—The prognosis depends upon the age and strength of the patient, the severity of the spasm, and the presence of particular complications. In simple Hooping-cough occurring in childhood, with moderate spasm and with an average state of health, it may almost always be regarded as favourable, and the disease may be expected to leave no ill effects behind. When it occurs in an infant under 4 months old, it is very liable to induce head symptoms, unless the attack be a very mild one; and when occurring in adult life, it is more likely to lead to chest complications and to leave permanent changes in the structure of the lungs.

When the paroxysms are unusually prolonged and the spasm very severe, complications are almost sure to arise; and if the child be feeble, the prognosis must be given with caution.

The complication of bronchitis renders the prognosis unfavourable only in proportion to its severity. When it occurs in a moderate degree and without inducing much dyspnœa in the intervals between the paroxysms, and at a season of the year when the temperature is not very low (the deaths from both Hooping-cough and bronchitis are inversely as the temperature), the gravity of the case is not seriously increased; but when it involves both lungs, and is attended by much dyspnœa and increased lividity of the countenance, the prognosis becomes unfavourable. Extension of the inflammatory condition to the substance of the lung adds much to the danger, from the fact that broncho-pneumonia is usually less amenable to treatment than bronchitis alone.

When emphysema and enlarged bronchi have already occurred, or when it is probable from the severity of the spasm that they will occur, permanent dyspnœa to a greater or less extent may be apprehended.

The occurrence of head symptoms attended by convulsions or paralysis must always render the prognosis unfavourable; but with convulsions not due to hydrocephalus, many cases recover.

The cases in which disordered secretions and intermittent fever occur will certainly be protracted, and may leave an enfeebled state of system from which the patient will not entirely recover, although life may be continued for many years. It is probably a less fatal complication than the others referred to, but it causes much anxiety both to the physician and the friends of the patient.

When extreme exhaustion is present without evident cause, the prognosis should be a guarded one.

It is a fact which is not sufficiently appreciated, that whilst so very large a proportion of cases of Hooping-cough recover, only six other diseases in the London district during the ten years from 1844 to 1853 inclusive, were more fatal than Hooping-cough; viz. phthisis, pneumonia, bronchitis, typhus, convulsions, and scarlatina.[1] Hence at the commencement of any attack of the disease it is well to speak of the future with caution.

Treatment.—There are but few diseases in which so many remedies have been employed as in Hooping-cough, and still fewer in which so much has been confidently asserted of remedies which differ much from each other. This is owing probably to the fact that the disease, running a more or less clearly defined course, usually subsides either with or without medical treatment, and with or in spite of whatever remedies may have been employed, and also that many remedies diverse in name and appearance are closely allied in their modes of action upon the system.

It is highly probable that many have been recommended on empirical grounds only; but others have been intended to effect one of the following objects: namely, to abate inflammatory action, to promote expectoration and the elimination of the supposed morbid poison, to diminish the bronchial secretion, and to allay the spasm, directly or indirectly, through the invigoration of the general system.

Those to abate inflammatory action have been leeches and antimony; to promote expectoration, antimony, ipecacuanha, squills, and other emetics; to lessen secretion, alum and zinc; to allay spasm directly, hydrocyanic acid, conium, hyoscyamus, belladonna, opium and morphia, musk, valerian, ether, and chloric ether, with various liniments applied to the chest and back, and a strong solution of nitrate of silver to the throat; to allay spasm indirectly, iron, zinc, copper, silver, and other metallic salts, mineral acids, with quinine, and other vegetable tonics, and change of air. Some were probably expected to have a specific action, as tar-water and tar inhalations, the air in the neighbourhood of gas-works, salts of lead and cantharides.

Simple Hooping-cough.—In the treatment of an uncomplicated case of Hooping-cough, the chief, if not the sole, aim should be to allay the spasm, and thus to prevent complications which result from it, and reduce the disease to a common cough.

As a preliminary step it is needful to regulate the functions of the chylopoietic viscera, to prevent or remove fæcal accumulation, to promote a proper secretion of bile, to prevent the occurrence of an excess of acid in the secretions of the stomach and œsophagus, and to prohibit the use of indigestible or irritating food. For these purposes, castor oil, or carbonate of magnesia, or Dinneford's or Murray's soluble magnesia, should be given daily or every second day, until the necessity for their use has ceased; and an occasional dose of one or two grains of hydrag. c. cretâ may be given at bed-time. The state of the nurse's milk should be examined in the case of suckling infants, and at a later period the food should be restricted as far as possible to cooked milk, and given alone or in puddings. Two or three pints of milk may be taken daily, but in such a manner that the quantity given at a time shall not exceed one quarter of a pint, and the intervals between the supplies be short. Bread should be rarely if ever given, except when cooked with milk. The use of vegetables should be greatly restricted. Meat in small quantities, and cut into very small pieces, may be given to a child of three years of age and upwards; and eggs made into puddings, and beef-tea, may be allowed at any age if there be a deficiency in the supply of milk.

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[1] Medico-Chirurgical Transactions, 1854, p. 230.

In the general management of the child the body should be kept properly warm by clothing, and the atmospheric air should not have a lower temperature than 64° either by night or day; and so long as the aim is to allay the spasm, the patient should be kept absolutely quiet both in mind and body, or as quiet as may be possible.

Such being premised, the next step depends upon the view which is taken as to the possibility of shortening the course of the disease. Some of the most eminent physicians of the present day are of opinion that it will run its course, and that the duty of the physician is to conduct it evenly and safely to the end.[1] With this view it is only possible to use palliative remedies, such as small doses of ipecacuanha, with or without rhubarb; but if a belief is entertained that the progress of the disease is amenable to treatment, the proper course is to select that sedative, narcotic, or antispasmodic agent, the action of which is the most uniform, and the dose capable of proper regulation; and the aim should be to administer it with the frequency and dose which will allay the spasm and *keep it under control* without interfering materially with any vital action.

Hydrocyanic acid has been strongly recommended by, amongst others, Drs. Granville, Hamilton Roe, Atlee, Elliotson, and West; and of these Dr. Roe has given the most detailed and judicious directions. He prescribes three-quarters of a minim of Scheele's strength to an infant, 1♏ to a child aged three years, and 1½ to 2♏ to one aged ten or twelve years, to be given every three or four hours, or even more frequently when the effect of the previous dose has abated. He has given 1½♏ every quarter of an hour for twelve hours to a girl aged ten years. He attaches great value to its action in reducing fever—a state of system which in simple cases rarely exists; but he also affirms that it will cure simple Hooping-cough quickly, or at any rate abridge its duration. Laurel-water, which contains hydrocyanic acid, has been given in doses of 6♏ to children and 30♏ to adults every two or three hours. Belladonna has been strongly recommended by Boerhaave, Hufeland, Guersant, Trousseau, Williams, Jackson, Churchill, G. A. Rees, and others. The dose of the extract recommended varies from $\frac{1}{12}$ to $\frac{1}{4}$ grain for a child aged two years, and $1\frac{1}{2}$ grain for a child aged four years and upwards; and Dr. Williams, bearing in mind its action upon the iris, and the desirability of diminishing the irritability of the bronchial and laryngeal muscles, regards the action upon the former as the measure of the action upon the latter, and takes it as his guide.

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[1] Sir Thomas Watson's Lectures.

Conium was introduced as a specific remedy for this disease, and has received the support of many distinguished physicians. The dose of the extract most commonly employed is $\frac{1}{40}$ of a grain for a child aged four months, $\frac{1}{20}$ to $\frac{1}{16}$ for one aged one year, and $\frac{1}{10}$ to 1 grain for older children, repeated every six hours. In the use of this drug, as in that of hyoscyamus and belladonna, it is customary to add small doses of ipecacuanha, and some physicians, as Guersant, combine it with the oxide of zinc or other metallic salts.

Tincture of lobelia is now given by Dr. Sidney Ringer with very good effect. The dose is 5♏ for a child æt. one or two years, increasing to 10♏ as the age advances to ten years, and is repeated every hour.

The preparations of opium which have been most generally used are the tincture of opium, and salts of morphia; but Battley's sedative solution and codeia have also been much commended. In a paper published in the Edinburgh Medical Journal, May 1856, we took occasion to express the opinion that morphia was the best remedy in this disease, since it is more certain and uniform in its action than belladonna, conium, hyoscyamus, and digitalis, and exerts a less injurious effect upon the sensorium and the bowels than the tincture of opium. It has already been pointed out that Drs. Roe and Williams, when administering hydrocyanic acid and belladonna, understood the importance of giving them in doses sufficient to allay the spasm; but in the paper referred to we endeavoured to explain that an essential part of the treatment was to carry this influence just so far as to be evident to an observer, and to maintain it during the period of treatment. With the view that the essential character of Hooping-cough, and that which leads to dangerous complications, is the spasm, and that the removal of the spasm should be the object of the physician, our aim was to cause the slightest oppression of the sensorium as a measure of the required effect of the drug, and to maintain it from three to six days. With children under one year of age the dose of the hydrochlorate or the acetate of morphia should be $\frac{1}{64}$ of a grain repeated every four hours; with children between one and three years of age, $\frac{1}{48}$ to $\frac{1}{32}$ of a grain; and with those yet older, $\frac{1}{32}$ to $\frac{1}{20}$ of a grain. The dose selected should be repeated three or four times; and if no perceptible drowsiness should be induced, it should be increased a step and repeated in like manner, and again increased if necessary until the dose has been ascertained which produces a very slight oppression of the sensorium. The aim must then be to maintain this effect by repeating the same dose, or by further increasing it from time to time. The cases of simple Hooping-cough are extremely few in which *slight* drowsiness has been produced and uniformly maintained for three or four days without the spasm having subsided, and the cough nearly reduced to that of a common cough. The plan, when intelligently carried out, has been most successful in our hands, and in those of Dr. Müller in Germany, as well as in the practice of many in England. It is rarely necessary to add any other remedy; but in certain cases the exhibition of carbonate of soda in addition to the morphia has further lessened the irritability of the larynx and promoted expectoration.

A very favourite combination is that recommended by Dr. Pearson, consisting of one drop of tinct. opii, five drops of vin. ipecac., and two grains of carbonate of soda, to be given every four hours after the operation of an emetic.

Dr. Eben Watson has proposed an admirable plan of treatment in the application of a strong solution of nitrate of silver to the larynx, by which the spasm is quickly relieved. The strength should be twenty grains to the ounce, and the solution applied not only to the tonsils and uvula but to the back part of the fauces, and if possible to the seat of the epiglottis. This is more difficult to effect in very young children, but in children of eight years of age and in adults it may be readily performed by depressing the back of the tongue until the free edge of the epiglottis is seen, and sweeping the pharynx with the camel's-hair brush or the mop charged with the solution. The application should be repeated every second day, and the spasm may be expected to subside in less than a week.

The external application, to the chest and between the shoulders, of belladonna plasters and of liniments containing belladonna, opium, or oil of amber, has been much employed with advantage; but the effects of belladonna must be watched, and dilatation of the pupil restrained within very moderate limits.

The inhalation of ether and chloroform, when diluted with air with the aid of a proper instrument, is often of great advantage in cases where the spasm is very severe and the patient not very young. It should never be carried so far as to induce anæsthesia.

The employment of metallic salts in the treatment of this disease has been very general and extensive, and those of arsenic, copper, and silver have been regarded as specifics. The proper dose of liq. arsenicalis is one drop daily, divided into four doses, for an infant, and one drop twice or thrice a day for a child of five years of age and upwards, and it may be given with water or decoction of cinchona. Carbonate of iron is prescribed by Dr. Graves after recovery from inflammatory symptoms: but others give the sulphate, and employ it in the early stage of the disease. In Dr. Rees's opinion it is particularly adapted to those of a strumous diathesis. Zinc has been given both to lessen the secretion from the mucous membrane and to allay the spasm. The proper dose of the oxide is one grain three or four times daily for a child aged one year, and two grains four to six times daily for those of five years of age and upwards. Acetate of lead has been particularly recommended by Dr. Rees, and he affirms that $\frac{1}{4}$ grain given every six hours removes the spasm on the first day of its exhibition.

Sulphuric, hydrochloric, and nitric acids have all been given in this disease with advantage when no inflammatory complication existed. Of these nitric acid has received the most recent support, and has been given in remarkably large doses. To a tumblerful of thin syrup as much dilute nitric acid is added as will render it as sour as lemon-juice, and of this a child under one year of age may take a dessert-spoonful every hour, and an adult the whole tumblerful in three or four hours. So much as ℈ij to ʒj of the dilute acid is given to a patient ten years of age and upwards, and 10♏ to a very young infant, when well sweetened with honey or sugar. No evil results are said to have followed, and the beneficial effect has been, it is affirmed, proportioned to the dose. Injury to the teeth is averted by using a gargle with carbonate of soda after each dose. This plan of treatment has received the approbation of Dr. Gibb, and its mode of action is presumed to be that of a tonic, sedative and antiseptic.

Change of air is a very popular remedy, and it is affirmed that it matters little whether it be to a purer or to a less pure atmosphere, but above all others the air in the vicinity of gas-works and lime-kilns has been, even very recently, commended. Except upon empirical grounds, and in the absence of sufficient proof of the benefit alleged, we can advise only that change which country air may offer to children living in towns; and even this exerts no marked influence in otherwise healthy children until the period of recovery from the exhaustion which follows the disease.

Alum and tannin have been much commended with a view to restrain the secretions and to allay spasm. The former, when given in the nervous stage of the disease, was exceedingly efficient in the hands of Dr. Golding Bird. His prescription was: alum twenty-five grains, extract of conium twelve grains, with syrup and dill-water to make a three-ounce mixture, of which a dessert-spoonful was given for a dose to a child two or three years of age. Tannin is given in doses of one-sixth to three-fourths of a grain, and even to three grains, combined with hyoscyamic or benzoic acid every two hours.

Alkalies, as carbonate of potash or soda, sulphuret of potass, liquor potassæ and liquor ammoniæ, are oftentimes of value, when conjoined with other remedies, in rendering the bronchial secretion less tenacious, and in relieving disordered bowels.

Nux vomica and strychnine have been given with advantage in the stage of spasm. Certain antispasmodics, as musk, valerian, and ether, are of value, but they do not so immediately and certainly relieve the spasm as narcotics and sedatives administered so as to slightly affect the sensorium. They are more particularly suited to the stage of recovery from the spasm.

Emetics may be employed with advantage in those simple cases of Hooping-cough in which there is unusual difficulty in removing the secretion from the bronchi, whilst at the same time it is excessive in quantity and impedes respiration. But with the view which we entertain of the nature and treatment of this disease we do not think that emetics should be the chief remedies employed.

Such are the remedies which have been and may be properly employed in simple cases of Hooping-cough—hydrocyanic acid, belladonna, conium, morphia, arsenic, zinc, alum, nitrate of silver, hydrochloric and nitric acids,—all affirmed at different periods to be specifics, or, at the least, sure and speedy remedies for this disease. With the weight of testimony in their favour we cannot deny that they have been and are very valuable agents; and if we have given the preference to one of them, it is because by it we may the most readily and safely induce that gentle oppression of the sensorium by which the spasm subsides. The mode of administration is as essential a part of the treatment as the use of the drug itself, and the plan will succeed only in intelligent and careful hands.

Complicated Hooping-cough.—As the most frequent complication is that of bronchitis, care should be taken to watch the earliest indications of its approach. When it exists, the use of metallic, vegetable, and mineral tonics and astringents should be discontinued, and antimony or ipecacuanha in small doses should be added to the narcotic or sedative in use. If there be much febrile action, the patient should be placed in a warm-water bath up to the neck every night or every second night, and spt. eth. nit. with liq. ammon. acet. added to the medicine. With much oppression of the respiration and difficulty in removing the secretion, the occasional use of an emetic and the constant use of alkalies will be proper, and counter-irritation of the chest by blisters, turpentine, or mustard should be effected. If there are evidences of the extension of the disease to the parenchyma of the lung or to the pleura, the application of leeches and cupping at the root of the lung or over a painful part may be of great service; but the use of mustard or other rubefacients between the shoulders will usually afford the greatest relief. Even during this period it will also be needful to sustain the vital powers by good beef-tea, and as far as possible by milk, given in very small quantities at a time; and great care should be taken not to carry the antiphlogistic treatment so far as to lower the vital powers and induce disgust for food.

When convulsions occur, the first duty should be to ascertain if the teeth cause irritation, or if there be irritating matters accumulated within the stomach and bowels. If the former, the gums, when tender, should be lanced; and if the latter, the bowels should be freely evacuated and the character of the evacuations carefully watched. At the same time the patient should be supplied with beef-tea and milk. When hydrocephalus occurs, with or without convulsions, the treatment of the case must centre in that complication, and be such as will be elsewhere advised in this work. With heat of skin and head it will be proper to apply cooling lotions and even ice; and should the disease be active, leeches to the base of the head should be early used. The free exhibition of mercury in the form of calomel in small doses, or of hyd. c. cretâ, must be persisted in when effusion has been diagnosed. As the bowels are usually constipated in this condition, mercurials do not readily purge, and care should be taken to induce a sufficient action. Moreover, a state of inanition from want of food must not be permitted.

In the complication with remittent fever care should be taken to evacuate the bowels and to correct the secretions, in addition to the use of those remedies referred to under that disease in another part of this work. The cough, which is also then unusually distressing, will require the frequent exhibition of morphia, syrup of poppies, and of tolu. As soon as possible the patient should have a change of air.

When extreme exhaustion is the chief complication, it will become necessary to administer nitric acid with bark, or some of the metallic tonics with quassia, and to supply wine-and-water in small doses every three hours. Much care should also be taken to induce the patient to take food, in small quantities, made from milk, eggs, jelly, and meat every two hours; and if neither the exhaustion be extreme nor the atmospheric temperature low, to obtain change of air and a moderate degree of exposure to it.

The recovery from Hooping-cough always demands the use of tonics, of abundant animal food, and of change of air, and possibly also of wine administered in small doses. Sea-voyaging is then of great service.

CROUP.

By William Squire, L.R.C.P. Lond.

Definition.—An inflammation of the larynx and trachea in children, commencing in the air-passages and often extending into the bronchi. It induces thickening of the mucous membrane and an altered secretion which may become either membraniform or purulent. There is frequent, sharp, harsh, ringing cough; difficult breathing, with loud, shrill inspiratory sound; altered voice, at first hoarse, afterwards whispering, or extinct; fever, loss of appetite, thirst, and little or no difficulty of swallowing.

Synonyms.—Cynanche Trachealis, Cullen; Suffocatio Stridula, Home; Angina Inflammatoria Infantum, Russell; Acute Asthma of Children, Millar; Cynanche Stridula, Crawford; Angina Trachealis, Johnstone; Angina Polyposa seu Membranacea, Michaelis; Hives, Benjamin Rush; Cynanche Laryngea, Dick; Angina Membranacea, Goelis; Tracheitis Infantum, Albers; Laryngo-tracheitis, Blaud; Laryngite striduleuse, Guersant; Spasmodic Laryngitis, Charles Wilson.

Name.—Croup and Roup (hreopan, Anglo-Saxon, *Clamare*) were the names popularly applied to the disease when it was first investigated by Home; at the commencement of the present century they were equally in use in the neighbourhood of Edinburgh. The latter once had a wider range, having been used by Knox as a verb signifying to cry hoarsely, and Burns has "roupet" in the sense of hoarse as from a cold;[1] since then, however, it has disappeared from our literature, and Croup, which before Home's inquiry was as strange to England as to the rest of Europe, has been the world-wide designation of the characteristic group of symptoms attending impediment to the entrance of air into the windpipe. It is somewhat remarkable that even in those countries where the disease is not infrequent, it is rarely distinguished by a proper name; Bräune in Germany, and Hives in America, being the only examples of which I am aware. Strypsiucka in Sweden has more the signification of quinsy or suffocation, and was not popularly applied to this disease when Rosen wrote; our own "strangles," "closing," "chock," or "stuffing," have neither been generally used nor definitely applied.

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[1] John Jamieson, D.D. an Etymological Dictionary of the Scottish Language. Edinburgh. 4to. 1808.

The Mœso-gothic hrop-jan is here given as the root of many words, signifying outcry, as croak, rout, hoop; also of the Teutonic roep-en and the Icelandic hroop. In the northern counties of England roopy and ropy are still used for hoarse, and the latter word is sometimes heard in the southern counties in the same sense.

HISTORY.—How large a share Croup has had in the various anginas or cynanches enumerated in the earlier stages of the history of medicine, it is impossible to define; in the subdivision of those terms by Boerhaave, there is evident intention of including it, and there is evidence that it was so included by our English physicians, from Sydenham to Mead. As Home remarks: "Probably it has existed, more or less, in all ages, for the same productive causes must have operated formerly as they do at present."

There are other systematic names under which cases of this disease have also been included, such as the "suffocative catarrh" of Ettmüller, and the "tussis convulsiva puerorum" of Willis; though the first of these names is now restricted to capillary bronchitis, and the second to hooping-cough, yet there is a clear reference to Croup in the pages of Ettmüller. The first evidence of Croup noticed by Baillou was in an epidemic of hooping-cough in Paris, 1576,[1] and the first mention of it by name in this country begins with the distinction between it and hooping-cough drawn by Dr. Patrick Blair,[2] in a letter to Dr. Mead, dated Cowpar of Angus, July 6th, 1713, wherein he says: "The tussis convulsiva, or chink-cough, is also some years epidemical and becomes universal among children; as is a certain distemper with us called the Croops, with this variety, that whereas the chink-cough increases gradually, is of long continuance, seizes in paroxysmes, and the patient is well in the interval; this convulsion of the larinx, as it begins so it continues, so violently that unless the child be relieved in a few hours 'tis carried off within twenty-four, or at most forty-eight hours. When they are seized they have a terrible snorting at the nose and squeaking in the throat, without the least minute of free breathing, and that of a sudden, when perhaps the child was but a little time before healthful and well. The most immediate cure is instant bleeding at the jugular, either by the lancet or leeches; when the most urgent symptoms are gone, then emetics or the like are administered at discretion."

The distinction is not always attended to, as even Huxham,[3] writing "de pertussi puerorum," speaks of an acrid humour sometimes attacking the larynx.

Dr. Russell[4] gives us an account of the disease as observed by him in connexion with the epidemic of malignant angina then prevalent, from which, however, he is careful to distinguish it. He says:—

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[1] Baillou, Epid. Ephem. Lib. ii. pp. 197 and 201.

[2] Observations in the Practice of Physic, etc. London, 1718.

[3] Huxham, Obs. de Aëre et Morbis Epidem. Lond. 8vo. 1793. P. 77.

[4] Russell, Dr. Richard. Œconomia Naturæ in Morbis acutis et Chron. Glandularum. 8vo. Lond. 1755. P. 72.

"I have observed it is most apt to seize children from two years old to eight or ten, but chiefly the younger sort." He details the leading symptoms, and remarks that the whole "fistula pulmonalis" becomes inflamed.

Home's essay[1] is founded on the observation of cases where no epidemic complications prevailed. In a careful and most philosophical inquiry into the causes of the symptoms before him, he determined their dependence on the pathological changes in the larynx and trachea, and regarded the disease as an acute inflammation.

Millar,[2] who practised at the same time in the south of Scotland, and had similar cases of Croup under his observation, remarks upon, but gives undue prominence to, the spasmodic element in the paroxysm of the disease. Further attention is called to Millar's views by the publication in England of a letter from Dr. Rush,[3] of Philadelphia, and a discussion commenced, which has continued to our own day, in which the true nature of Millar's cases is not always remembered.

The latter half of the last century and the beginning of the present are remarkable for the numerous outbreaks of epidemic angina recorded in different countries and places. In the midst of an epidemic at Cremona, Ghizi had described the case of a child dying of laryngeal complication, and attempted to set up a distinction between it and the pharyngeal form of the epidemic. The cases recorded by Starr, in Cornwall, occur in his description of an epidemic of this kind, and it is probable that Russell's were not wholly isolated. Home, careful lest the distinct inflammatory disease which he had constituted should be confounded with an epidemic disorder of so different a nature, drew a distinction at the very commencement of his inquiry between his own observations and those recorded by Russell; yet fresh outbreaks of the epidemic, its liability to spread to the air-passages, and its severity towards children tended to their confusion.

One effort to avert the fatal mistake that ensued—and it is the only one—is recorded in the treatise of Dr. Johnstone the younger, of Kidderminster.[4] He quotes from Home the "two very different situations of the suffocatio stridula; the former more inflammatory and less dangerous; the latter less inflammatory and highly dangerous: in the former the pulse is generally strong, the face red, drought great, and they agree with evacuations; in the latter the pulse is very quick and soft, great weakness, tongue moist, less drought, great anxiety, and evacuations hasten death." Dr. Johnstone contends that these are not merely two stages of the same disease, but that the latter applies to that complication of the epidemic which has been observed in all its records from the earliest times, and that it was occasioned by the same epidemic cause, and required the same sustaining plan of treatment that his father adopted.

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[1] Home, Francis, M.D. &c. An Enquiry into the Nature, Cause, and Cure of Croup. 8vo. Edin. 1765.

[2] Millar, Observations on the Asthma and Hooping-Cough. 8vo. London, 1796.

[3] Rush, On the Spasmodic Asthma of Children. 8vo. London, 1770.

[4] Johnstone, J. M.D. A Treatise on the Malignant Angina, to which are added some Remarks on the Angina Trachealis. 8vo. Worcester, 1779.

Unfortunately, though argued with learning and experience, these views did not prevail; the name of Croup was applied to the epidemic complication, and the treatment laid down by Home for the one disease was very energetically employed against the other. The divergence of opinion tended to stimulate the collection of facts bearing on the subject. The accounts of epidemic Croup, though generally referring to the disease now known as diphtheria, doubtless comprehend some cases of simple Croup; some of our own accounts of Croup probably include cases of diphtheria. It was to illustrate the tracheal complication of the epidemic that both the great *concours* on Croup were instituted at Paris; yet the prize essays of MM. Vieusseux and Jurine, of Geneva, and of Albers, of Bremen, are among the most valuable contributions to our knowledge of Croup as an independent disease; and, though the tendency in France has since been to restrict the term Croup to one of the accidents of diphtheria, yet the opposite view has in that country been maintained with great ability by MM. Bricheteau, Desruelles, Emangard, and especially in the valuable original work of M. Blaud, of Beaucaire.[1] A similar controversy, arising under conditions more allied to those of our own country, has been continued in Northern Germany since the time of Wichman of Hanover, starting from a line of distinction being drawn between its spasmodic or inflammatory nature; the treatise of Goelis of Vienna[2] is, however, sufficiently comprehensive. In America the "Observations on Cynanche Trachealis," published in the first volume of "Medical Inquiries and Observations," by Dr. Benjamin Rush, will ever stand as one of the clearest and most practical accounts of the disease. In our own country we have the careful study of Cheyne,[3] enriched, as it is, by the admirable pathological drawings from the hand of Sir C. Bell: it forms a worthy sequel to the work of Home, conceived and executed in the same spirit, from observations made in the same locality at no great distance of time. We have also the matured experience of the same author, gained in Dublin and its neighbourhood, published thirty years later in the "Encyclopædia of Practical Medicine." Dr. Charles Wilson of Edinburgh has published, in the *Edinburgh Journal of Medicine* for 1855–56, a philosophical review of the whole subject.

ETIOLOGY.—The collection of facts which go to make up our history of Croup is sufficiently extensive; but, besides the uncertainty as to their true bearing which we see in some of them, others are drawn from too limited an area, or considered in too restricted a relation to come before us in their true value. I have therefore availed myself of the kind permission of Dr. Farr, to consult the careful reports prepared under his direction in the office of the Registrar-General for England. These reports extend over a period of twenty-five years; they contain particulars of nearly 95,000 deaths from Croup; and therein the locality of occurrence, the season, the sex, and age at the time of death, are readily investigated. I have also referred to reports for Scotland, extending over seven years and including 6,982 deaths registered as Croup.

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[1] Nouvelles Recherches sur la Laryngo-Trachéite. Paris. 8vo. 1823.

[2] De Rite Cognoscenda et Sananda Angina Membranacea. 8vo. Vienna.

[3] Essays on the Diseases of Children. Essay II. Cynanche Trachealis. Edin. 4to. 1801.

Croup is specially a disease of childhood, occurring most frequently from the first to the seventh year, and rarely happening after the tenth. In a hundred deaths from Croup, we may estimate 13 as occurring in the first year of life, 25 in the second, 22 in the third, 16 in the fourth, 11 in the fifth, and 12·3 in the succeeding five years, while the deaths beyond ten years of age may be represented by 0·7, the remaining fraction. The proportion of deaths from Croup to one hundred deaths from all causes, registered at each age, is 0·65 in the first year, 3·25 in the second, 6·5 in the third, 8·0 in the fourth, 7·0 in the fifth, and 3·5 for the five following years together. The proportion for the first and second year of life would be raised to above 1 and 4 respectively, if the deaths at these ages registered under the head of Laryngitis were included; some considerations, hereafter to be given, tend to raise the proportion for the first and second years and to diminish the already small proportion registered as occurring beyond ten years of age. In Scotland, the proportion registered for the first year is 1·5; something over 1 per cent. for the first six months, and for the second six months exceeding 2 per cent.; though the actual number dying from this cause in the first half-year of life is but little below, and in some years has exceeded, the number of deaths in the second. The annual average number of deaths from Croup in England is near upon four thousand; and, though this number is somewhat below the returns for the majority of the last ten years, and in excess of the greater number of any of the preceding ten years, yet the proportion to deaths from other causes has always been very nearly one in the hundred, somewhat above this for the last ten years, a little below it for the preceding ten, again above this proportion for all the preceding years that are registered; the first of these years, 1838, being as high as 1·30. The lowest proportion was 0·869 in the year 1853; the high proportion of 1·335 occurs for the first time in the year 1850; the highest proportion reached is 1·40 in the exceptional year 1858. In Scotland the proportion to other deaths is from 1·5 to 1·8 per cent.; the annual number of deaths is close upon one thousand. Dr. Burke, of Dublin, kindly provided me with the results of the first complete registration for Ireland, which shows that in the year 1864 the whole number of deaths from Croup was 1,926, and the proportion to deaths from other causes 2·05.

More boys than girls die of Croup; this fact is obvious over whatever period or district our inquiries extend: the difference is striking, and by frequent notice has been brought more prominently forward than the corresponding fact in the history of some other diseases chiefly fatal in childhood. More boys than girls are born, in a proportion somewhat greater than one in every fifty children, or, to give the result of a very extended examination,[1] there are 511·75 males and 488·25 females in every 1,000 births; it appears that of this number 83·71 males and 65·74 females die within the first year, after which the death ratio of the two sexes for the next ten years is nearly equal; still there are a larger number of males than of females living at this period, and the deaths of females from all causes are to those of males as 87 to 100 in the first five years, or as 88 to 100 in the first ten years: now the deaths from Croup are so nearly in this proportion, and of late years have so often shown a difference so much less than this, that a doubt might be entertained as to whether any difference in the liability of the sexes really existed. A comparison between the deaths from all causes of each sex for each year, with the deaths from Croup at each year, sex with sex, shows a difference of excess on the side of the males so constant that it is rare to meet with an exception, but at the same time so slight that it can only be considered a characteristic of the disease in the aggregate, corresponding with the results of pneumonia and tubercular meningitis, rather than with the more characteristic zymotic diseases, and contrasting with those of diphtheria and hooping-cough, where the excess of deaths is greatly on the side of the females. Some of the results of the preceding inquiry are brought together in the following table:—

Deaths from Croup at each year of age.	1st Year.	2d Year.	3d Year.	4th Year.	5th Year.	5th to 10th Year.	All ages beyond.
To 100 deaths from Croup.	13·	25·	22·	16·	11·	12·3	0·7
To 100 deaths from all causes.	0·65	3·25	6·5	8·3	7·5	3·5	
Males.	0·7	3·5	6·7	8·5	7·7	3·7	
Females.	0·6	3·0	6·3	8·1	7·3	3·4	

The influence of climate upon Croup is generally admitted. Cases considered trivial in some parts of France are frequently fatal in Northern Germany, and what in our variable climate excites alarm is regarded with reasonable hopefulness on the continents both of Europe and America. A combination of cold and moisture with rapid alternations of temperature, together with some endemic or epidemic influence, has to be admitted. In South America, Buenos Ayres, with its large river, affords frequent instances of Croup; and in some of the large towns of Australia, with their defective sanitary arrangements and large infantile mortality, Croup was not unknown years before the first appearance of diphtheria. The high mortality in Scotland is not greatest in its most northern extremity; the mortality from Croup in the northern counties of England is generally over 1 per cent.; but this is equalled and often exceeded in the warm south-western promontory of Cornwall, with Devonshire and Somerset. The western shores of England, receiving the Atlantic moisture, show a higher mortality from this disease than the eastern. South Wales has the high rate of 1·5 per cent. This is not so much owing to its mountainous interior, as to its large mining population and the defective sanitary state of its large towns. The highest rate for England is in the populous districts of Lancashire and Cheshire; and here there can be no doubt that a dense town population, the children specially living under defective sanitary conditions, causes Croup to be particularly fatal. That it is not merely the combination of cold and moisture, may be shown by the returns from the south-western corner of Scotland, Wigton and Dumfries, the latter conterminous with our Northumberland and Cumberland, and with a rainfall[1] exceeding that of any part of England. Though the temperature is sometimes very low, it is more equable than that of many parts of our island, and the mortality from Croup, generally below 1 per cent., is sometimes as low as 0·5. The classical Croup districts of Scotland still retain their pre-eminence; they are not confined to the west coast of Scotland: the eastern coast is deeply indented by the sea, and not only do deep valleys of clay extend from these firths, but their shelving shores leave a great expanse of ooze uncovered at every tide; and during the easterly winds, which here prevail for three months of the year with great bitterness, the characteristic cases of Scotch Croup occur. This part of Scotland forms an isthmus, only thirty miles in width, in the vicinity of which, and of the peninsula formed by the eastern firths, Croup is most fatal, the mortality often exceeding 2 per cent. As high a rate is found for Ireland, where imperfect drainage, unreclaimed bog, and a large expanse of inland water add to the influence of the Atlantic in causing a remarkable humidity of climate.

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[1] English Life Table, with an Introduction, by W. Farr, M.D. F.R.S. London, 1864. Table III. p. 24.

The influence of season is illustrated by the quarterly reports for London; an average of the ten years from 1844 to 1853 gives the number of deaths from Croup in each quarter as follows: first quarter, 95; second quarter, 81; third quarter, 68; fourth quarter, 92·5; the greatest fatality being in the winter and spring; the greatest variation is found in the second quarter; the third quarter has the lowest number, and shows the least variation; the fourth quarter generally shows a considerable increase on the third, or warm quarter of the year: this was less marked than usual in the year 1852; for while the deaths in the third quarter showed a tendency to increase, being as high as 74, there were only 76 in the fourth quarter, the unprecedented mildness of the season doubtless being the cause of this arrest; the two usually cold months of this quarter, November and December, averaging throughout a temperature 6° higher than had ever been known during the past eighty years. In the next year severe cold set in before the end of January; in February, the temperature was below the average on most days, there was snow every day; the second week in March was warm, the end of the month cold with snow; the summer was variable, cold, and wet; double the usual quantity of rain fell in July; there was fine weather in August only. Again, from October 21st to November 8th, the temperature rose to 5·3° above the average, at other times it had been below, and November and December were remarkable not only for low temperature, but also for a density of fog and depth of snow hardly ever exceeded in London. The mortality from Croup in each of the four quarters in this year was: first quarter, 93; second, 79; third, 72; fourth, 130; and 145 in the first quarter of the next year, 1854, the weather continuing to be cold. The weekly returns for this period show a correspondence between mortality from Croup and temperature. In the winter quarter of 1852, the weekly numbers for November are, 3, 5, 8, 5; in the corresponding weeks of 1853, they are, 7, 13, 8, 12; the high number occurring on fall of temperature in the second week of that month. In the previous March there was a rise of temperature in the second week: the weekly numbers for this month were, 15, 8, 2, 10; and in the next year, 1854, there are 21 deaths from Croup returned in one week in February. Cold, however, may determine the incidence, but not the prevalence of Croup. In most diseases of the respiratory organs, the greatest fatality is seen in the coldest seasons. The proportional mortality in this class mounts from 11 per cent. in 1852 to 13·5 in 1853, and hooping-cough from 1·8 to 2·3 per cent., while Croup decreases from 0·99 to 0·86 per cent.; the whole number of deaths from Croup being less in 1853 than it had been for several years.

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[1] At Wanlock Head, in Dumfriesshire, there was an estimated rainfall of 80 inches in the year 1861.

A further illustration of the influence of season and the relation between Croup, the diseases of the respiratory organs, and prevailing epidemics, is afforded by the years 1859, 1860, 1861. The quarterly returns of the mortality from Croup in London for these three years are as follow:—

	1st Quarter.	2d Quarter.	3d Quarter.	4th Quarter.
1859	132	103	80	81
1860	117	80	105	169
1861	236	190	170	252

In the first of these years the summer and autumn were fine and hot. June had a daily excess of 3° of temperature; in July the mean temperature was 68°, and on the 13th and 18th of that month the thermometer reached 93°; part of October and November was cold, but it was warm at Christmas. The next year, 1860, presents a remarkable contrast to this; a cold period commenced in June: of the three following months, Mr. Glaisher in his report[1] says, "The weather during the past quarter has been very remarkable for continued low temperature, frequent rain, large amount of cloud, little sunshine, and bad weather generally;" the winter that followed was one of the coldest on record, the thermometer being as low as 6° Fahr. in London on Dec. 6th-7th, and at Nottingham it fell to 8° below zero, or 40° below the freezing-point of water; a rapid thaw set in on Dec. 30th, and though there was severe cold in January, the remainder of the winter was more remarkable for rapid changes of temperature than for continued cold. In 1861 the spring was variable, but the summer and autumn unusually hot and dry: in June the thermometer was 82°, and on August 12th it was 89·5°; the years 1770, 1811, and 1831 only had as warm an October; with the exception of a cold week in November, the warm weather continued up till Christmas. In the cold season of 1860, diseases of the respiratory organs advanced from 13·7 to 16·4. Hooping-cough, however, did not increase, and laryngitis observed a considerable decrease. Croup also decreased from 1·29 to 1·05. During the high temperature of 1861 bronchitis decreased, hooping-cough was on the increase, laryngitis and Croup continued the same.

The London quarterly reports give similar evidence. Diseases of the respiratory organs increased from 15· to 20·; bronchitis, from 8· to 10·; pneumonia, from 5· to 6·7; laryngitis and Croup were 0·4 and 0·7 respectively in the warm season of 1861; when the former diseases were declining to their usual standard, the latter made their most rapid increase in London.

Laryngitis is also approximated to Croup in the time of year in which it is most fatal.

The proportional mortality in London from diseases of the respiratory organs is, for each quarter,—

Diseases of the Respiratory Organs	25·	15·	10·	20·
Bronchitis	15·	5·	9·	10·
Pneumonia	9·	6·	5·	8·
Laryngitis	0·6	0·5	0·3	0·4
Croup	0·9	1·0	0·6	0·8

There is a strong contrast between Croup and bronchitis as to the time of year at which each is most fatal; the difference is less marked between it and pneumonia, probably from a larger proportion of its victims being among the young; there is a close correspondence between laryngitis and Croup in this respect, and the table shows also the increased proportion in which they have latterly appeared in London.[2] Eighty-five per cent. of the mortality from laryngitis is among children; and in the great increase in the deaths from this cause during these three years, viz. from 260 in 1859 to 386 in 1861, there are 42 of the smaller number, and about 50 of the increased number of these deaths that occurred beyond the tenth year.

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[1] Remarks on the Weather during the Quarter ending Sept. 30, 1860, by James Glaisher, Esq. F.R.S., in Registrar-General's Report.

[2] The deaths from these causes in London for each quarter average for 1850-53: Croup, 92, 78, 62, 94; Laryngitis, 68, 61, 34, 43. For 1859-61: Croup, 161, 124, 115, 134; Laryngitis, 110, 74, 47, 68. Before 1845 it was not usual to separate infantile laryngitis from Croup. The average quarterly returns of deaths from laryngitis for the five years 1840-44 were 7, 9, 6, 10; for the subsequent five years, 47, 38, 25, 44; the returns under the head of Croup showing at the same time a diminution.

One of the influences bearing upon the mortality of Croup, is that of associated epidemics. In the year 1853, when deaths from croup were few, the mortality from diseases of the zymotic class was 20 per cent. or nearly at its lowest. Small-pox was on the decline; measles reduced to 1·1 per cent., having been 2·3 in 1851; and scarlatina, though less than it had been, was still 3·7 per cent. From this time an increase is observable in the number of deaths from Croup. In 1854 the mortality from measles was again over 2 per cent.; scarlatina was over 4 per cent. in this year and the next; and though in 1856 it fell to 3·6, and measles to 1·8, and epidemic diseases generally did not exceed one-fifth of the mortality, yet a new epidemic disease, allied to scarlatina and closely associated with Croup, was developing in England: many of its first victims were registered under this head; and Croup, which was 1·05 per cent. in 1855, rose to 1·34 and 1·27 per cent. in the two following years. The year 1857 was unusually hot;[1] 1858 was an epidemic year; diseases of this class constituted more than one-fourth of the general mortality; diphtheria had not been separated in the registers, and the returns under the head of Croup and scarlatina were increased, the one by a thousand, the other by more than ten thousand, and their proportional numbers to 1·4 and 6·8 respectively. In the next year Croup is 1·3 and scarlatina 4·5 per cent., diphtheria appearing as 2·2 per cent. In the next two years, 1860 and 1861, the mortality from epidemic diseases is reduced to less than one-fifth of the whole, scarlatina and measles are about 2 per cent., and diphtheria 1·25. Croup is again 1 per cent. In 1861 the number of deaths from diphtheria is at its lowest, that from Croup a little higher than in 1860. The increase was chiefly at the end of the year, and was almost confined to London and Lancashire; the deaths in this part of England were more by 2,000 in the last quarter of the year than in either of the two previous winter quarters; in Manchester they rose from 1,682 to 2,123, and other large towns in this district show a similar increase. In London the chief coincidence is that of the increase in the mortality from scarlatina from 467 in the third quarter to 1,145 in the last. In the corresponding quarter of 1860 it was 602. Hooping-cough and fever were on the increase in these districts, and from this time the commencement of an epidemic period may be dated.

The year 1862 was cold, wet, and unhealthy; the mortality from Croup is again 1·3 per cent.; from hooping-cough, 2·8; from other diseases of the respiratory organs, 15·6; scarlatina is increased to 3·4 and diphtheria is 1·13; zymotic diseases generally have increased to 21·2; the whole mortality during the year being very great.

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[1] "The temperature was 2° above the average of the preceding 17 years; the wind, instead of moving at the average rate of 110 miles a day, passed over London at the rate of 81 miles during the 53 weeks. The rainfall was 21·4 inches."—*Registrar-General's Twentieth Annual Report*, p. xliii.

It is to some general causes acting unfavourably upon the health of children, rather than to the influence of a particular epidemic, that these variations in numbers are to be attributed. Croup, indeed, seems to hold a place intermediate between diseases of the zymotic class and those of the respiratory organs.

Diseases of the zymotic class generally show a greater mortality among females than males. Croup differs from them as a class in this respect. Hooping-cough exemplifies this point of difference in the greatest degree; Measles, though often followed by Croup, shows no periodical coincidence with it, except in the time of year at which it is most prevalent, cold appearing to increase the fatality of both; Scarlatina is distinguished by being least fatal in the spring; while Small-pox is not modified in its violence by either season or climate. Whenever these diseases have increased there has been an increase, at least, in the fatality of Croup, and the same is noticeable with respect to Diphtheria. This differing from the preceding diseases in its liability to recurrence, and to some extent corresponding with Croup, is distinguished, as to these general characteristics, by showing a greater mortality among females; by being, when least epidemic, like scarlet fever, most fatal in the autumn and winter; and when most epidemic, like small-pox, by a progression independent of season and climate.

Croup differs from diseases of the respiratory organs in its periods of greatest mortality, and widely from some of this class as to prevalence at different periods of life; the whole class agrees with Croup in showing a greater mortality of males than of females, and the effects of climate and season are always obvious in both. One disease of this class, infantile laryngitis, has been specially commented upon, to set forth its contrast with the class, and its affinities to Croup, and to show by the intimate correspondence of the two in every particular by which they can be compared, that this form of disease should not be considered, from the absence perhaps of one anatomical character, as in any way different from Croup, nor hereafter be classed apart.

Croup has also some relations to diseases of the constitutional class. In the severer forms of local inflammations, individual susceptibility is concerned; and besides a predisposition, induced either by previous illness or by the causes examined, some constitutional infirmity may predispose, and it is noticeable that this source of disease is more apt to come out in the first decade of life among males, and just after that period in females. Croup affects children of certain families, and certain children of some families, more than others; it is said that those of a florid complexion are often more liable. We see in a family some suffer much under infantile disorders that affect others but slightly, without knowing why. Not only is the tendency to spasm hereditary, but to local congestion, at particular ages. Where there is no diathetic peculiarity, some aberration from health is a usual predisposing cause. There are other causes, such as sudden changes of dress, the impression of cold air after heating exercise even residence at the sea-side, which may influence the frequency, though not the fatality, of Croup. Children who have suffered an attack are specially liable to a recurrence on exposure to any of these causes, and the recurrent attack is not always the least severe.

SYMPTOMS.—The symptoms of Croup follow quickly upon the cause which excites them; the first indication is often mere hoarseness in the tone of voice or cry, the child is feverish, and either dull or fretful; is thirsty, and drinks without difficulty; the tongue has a white fur, and is red at the tip and edges; there is some heat and dryness of the skin, and a check to the secretions generally; an occasional short dry cough may be noticed, and a little harshness of breathing. The more characteristic symptoms generally come on at night; during the first sleep the cough is noticed to be sharp and harsh, with that peculiar croupy clang which, when once heard, is always easily recognised; this may be repeated at some intervals without rousing the child from sleep; the heat and dryness of the skin are now more marked, the pulse is frequent and strong, and the breathing loud and difficult, when some repeated clanging cough, with shrill-drawn breath, wakes the child in a fright struggling for breath. He starts up, is flushed and hot, the eyes staring, the conjunctivæ red, a hissing sound accompanies every inspiration, and is very marked and loud after the short dry sounding cough; it is evident that insufficient air enters the chest, although the respiratory efforts are great; the circulation is now also highly excited, the turgescence of the face and neck increases, and the colour deepens, the child puts its hand to its throat as if to remove obstruction, speech becomes impossible, and soon, as if in despair, muscular effort relaxes and air begins to enter the chest more freely.

The paroxysm may come on within a few hours of exposure, and sometimes, though rarely, before the usual symptoms of ingress have been noticed; it may begin at any hour, but most frequently at night, and is seldom delayed beyond thirty-six hours from the commencement of the illness; it may last but a few minutes, or be prolonged with varying intensity for more than an hour; its first accession is nearly always followed by a remission, more or less complete, sometimes so perfect that the most careful examination is required to ascertain the presence of the disease, and to prevent a fallacious confidence following too closely upon the first alarm. Its intensity also varies much; but however slight in degree, it occasions an acceleration of pulse, an increased heat and redness of the surface, especially of the face, also an injection of the conjunctival vessels not existing before, and which will probably subside if the paroxysm be not too soon repeated.

The temperature as taken in the axilla has been found at 100°, with the first premonitory symptoms; by the second day, or at night, it will rise to 102°, possibly to 103°, coincident perhaps with the earlier paroxysms, but not with the severer attacks which may follow: indeed the temperature is generally less on the third day, and will subside by the fifth unless pulmonary complication have arisen. A high temperature at the very onset may point to one of the exanthemata; its persistence to diphtheria.

It is important to consider whether the patient is being seen shortly after such an attack; for if seen before, or at some time after a first slight attack, there will be neither redness of conjunctiva nor coryza, and even though sneezing or some catarrhal symptoms have preceded, there will be no defluxion from the nose; the skin will be dry and harsh rather than hot; the urine will be found to be in small quantity and of a high colour, with no marked sediment. The pulse is quick and hard; the respiration is accelerated, but, though disturbed and somewhat oppressed or wheezing during sleep, it is not much altered in frequency; and, unless there be already some implication of the lung, or the presence of some other disease, it will not have a ratio of more than one to three, nor of less than one to four pulsations.

The most valuable indication of the presence of the disease, even in this early period, is drawn from the respiratory sounds and movements. The voice may attract attention, the cough will soon give the trumpet-note of alarm, but, without the impeded respiration and its physical signs, their indication is not conclusive; they may even be absent during some temporary lull in the symptoms, and then is the favourable moment for a careful auscultation. The inspiratory sound is prolonged, and, instead of the ordinary blowing murmur, there is a sibilant tubular sound, high in pitch and of a metallic quality, constituting a prolonged harsh stridor; the expiratory sound is also prolonged, but is low in pitch and harsh, the respiratory murmur is weak, especially in the anterior and upper part of the chest, and is masked by the tracheal siffle; this is very marked over the larger bronchi, but is not always enough to conceal the presence of a certain amount of mucous and sibilant rhonchus in some of the smaller bronchi posteriorly; there is no dulness on percussion, not even over spots where the murmur is altogether absent. The respiratory movement may also be noticed to be deficient in this stage, and when exaggerated during dyspnœa, to be inefficient; the supra-clavicular spaces are depressed during inspiration, and though the diaphragm may descend well, the intercostal spaces will not bulge, nor will the walls of the chest be fully expanded. The cough is sure not to be long quiet, and its short, dry, abrupt character attracts notice; it is not, strictly speaking, a hoarse cough, there is no deficiency in body of sound, and it is high in pitch; a shrill inspiration accompanies each effort; during the paroxysm the cough will be frequent, and it is then the sign most worthy of attention. Our further investigation of the state of the respiratory organs must at this time be limited to inspection of the front of the chest, and percussion at the back; when the attack is over, during sleep, or after vomiting, auscultation can be satisfactorily accomplished.

The condition of the whole extent of the body should now be examined, to remark the absence of spasmodic contraction of the thumbs or toes, the presence or absence of a rash on the skin, of œdema of the extremities or of the face and neck, and the degree of warmth and tone of colour of these parts as compared with the body generally; the last point is to be specially noted, so that any variation in the depth or tone of colour in these parts and in the face and lips may be readily appreciated. The sides of the neck are to be examined for enlarged glands; those at the outer border of the sterno-mastoid are always palpable, but it is important to note that the glands at the angle of the jaw are not enlarged. An early opportunity must be taken for a full and clear inspection of the inside of the mouth and throat. There may be some redness of the soft palate, sometimes œdema of the uvula; the pharynx will be either of a pallid red or of a brighter pink hue; there will be a remarkable absence of free secretion, and no speck of adherent exudation visible in any part of the pharynx or tonsils: some enlargement of the tonsils has been noticed, and if it be sufficient to press forward the anterior arch of the palate a slight irregularity of outline will be occasioned, but the membrane is continuous, of uniform colour, and smooth. Before this inspection is ended, the tongue should be sufficiently depressed to bring the epiglottis into view; the vivid redness and turgescence of its apex contrasts strongly with the surrounding textures, and indicates the condition of the subjacent orifice.

The disease attains its height by the end of the third day at the latest, but the intensity of the attack may hasten the stages of its advance, and death may occur within forty-eight hours of its commencement. The characteristics of this second period are high vascular excitement, and an ever-increasing difficulty of respiration; the cough is now almost incessant, or frequently recurring in shocks of convulsive violence; there is no free secretion; a little viscid phlegm, clear or muco-purulent, may sometimes be expelled, or an opaque mucus be seen in the lower part of the pharynx. Pain is complained of in the front of the larynx, or nearer the top of the sternum; the voice may become whispering or suppressed from the effort to speak being evidently painful; the stridulous inspirations are louder and more continuous, the laboured and sonorous breathing being audible at a distance; and now, though the thirst is great, deglutition is not always easy, in some cases, from imperfect closure of the glottis, the liquid provoking great dyspnœa; in others the urgency of the dyspnœa itself not permitting the effort. Even at this stage, if the attacks of dyspnœa have neither been too severe nor too frequent, and air sufficient to maintain life be yet admitted to the lungs, the pulse will steadily maintain its force and frequency; there will be great heat of surface and profuse perspiration, especially on the face and forehead, which parts will be of a bright red colour; the veins of the neck and temples may become distended and dark, and the face and lips at times purple, but if they quickly assume a brighter tint it may not be too late for the disease to take a favourable turn. The first evidence of this is a change in the character of the cough; it becomes lower in tone and less dry, not less in force nor much less in frequency, but becoming moist, and gradually effecting the expulsion of some thick semi-opaque mucus in which not unfrequently small whitish opaque flakes are discernible. At the same time the sibilant inspiration is neither so loud nor so persistent; it is still heard before each cough, and it will be audible during sleep or on first waking; the voice at times regains its natural quality, at others is only hoarse or dissonant in its higher tones; the accessions of dyspnœa are rare and less marked, the febrile excitement subsides, a more equable perspiration is maintained, the urine becomes abundant and frequently affords large deposits of urate of ammonia and sometimes of oxalate of lime; the soft palate, tonsils, and pharynx become paler and less tumid; a loose muco-purulent secretion is often seen in the gullet; the tongue is less red, less furred, and more moist; thirst diminishes, and appetite returns; the harsh tracheal siffle will have been replaced by some mucous râles, the normal respiratory murmur will be everywhere restored during this favourable progress, and, if uninterrupted, three days may suffice to establish convalescence. A persistence of irritability in the air-passages may greatly delay this, and a further extension of disease to the lung endanger it altogether. Auscultation here again becomes the only basis of confidence, as upon the subsidence of the more urgent signs, others, less obvious but not less important, may be discovered; it may be found that there are parts of the lung to which air is not admitted; that there is an accumulation of mucus in the bronchi, or an amount of bronchitis, with the development of subcrepitant rhonchus, that will seriously impede recovery. An extensive capillary bronchitis, or the existence of pneumonia, will not only be indicated by their special characters, and by the acceleration of the pulse-respiration ratio, but also by the general symptoms; the signs of laryngeal obstruction have diminished, but the respiration is as much embarrassed; there is less effort, but there is no relief; and the disease advances to its close as surely and even more hopelessly than if its advance had been unbroken.

The third stage is that of apnœa and rapidly advancing exhaustion: it may come on in the manner just described, but more frequently is the direct sequence of the more urgent symptoms of the second stage, which when about to lead to this result present some additional noteworthy particulars. The tracheal siffle is accompanied by "tremblotement," a laryngo-tracheal mucous râle with a tremulous character heard in both expiration and inspiration, or a click either constant or occasional may also be heard through the stethoscope; and whatever the character of the râle or siffle, it now becomes audible over the trachea in expiration. The voice is whispering or completely suppressed, the cough stifled, powerless, or altogether absent; it may, however, recur in some paroxysm of dyspnœa and afford temporary relief by the chance expulsion of some membranous shreds. The hand may at times be directed to the mouth or throat as if to remove some obstruction, but the paroxysms become more urgent and without remission, there is restless tossing of the body and limbs on the bed, consciousness is impaired, and voluntary power much diminished; the respiratory efforts may continue for a time to be violent, loud stridor marks both the expiration and inspiration; with the latter act the larynx is seen to be forcibly drawn towards the sternum, the supraclavicular and intercostal spaces sink, and though the abdomen descends, the epigastrium recedes; the head is thrown back, the lower jaw fixed, the mouth partly open, the alæ nasi dilated and depressed; the veins of the neck and temples are distended and dark, the eyes starting, and the face livid; the pulse becomes either too rapid or too weak to be counted, the temperature falls and the perspiration becomes cold and clammy, the neck and even the extremities may be swollen as well as the face, and assume a leaden colour, or the whole surface becomes of a marble-like pallor, the features are set, the eyes lose their expression, oscillate in the orbits, become distorted and fixed. Sometimes the whole body is bent backwards, and death has been known to occur at the moment of an inspiratory effort. Suffocation is every moment imminent, but frequently an apparent calm in the more violent symptoms precedes death, a gradually decreasing quantity of air is entering the lungs, the whole chest is flattened and much diminished in fulness and capacity; the countenance, though not livid, no longer retains its florid hue,—it becomes shrunken, dusky, or pallid; complete stupor sets in, the limbs become flaccid, the surface cold; the pulse is small, weak, and frequent; the eyes are dull and sunken; the respiration becomes gasping and irregular, the pulse intermitting, and both soon cease. The whole duration of the disease, advancing uninterruptedly to its fatal termination, rarely exceeds five days.

The division into the three stages of ingress, full development, and termination by apnœa, is an arbitrary one; the limits of each cannot be defined, nor is there any natural line of separation; whenever apnœa is commencing, the third stage has arrived, and this may be suddenly fatal, even though the first stage seems unaccomplished; so also in the second stage the signs of increasing obstruction in the trachea may be undeveloped, and yet the third stage have set in and be gradually advancing.

DIAGNOSIS.—Numerous causes affecting the glottis and larynx, modifying their special functions, and interfering with the entrance of air into the lungs, give rise to croupal symptoms that are to be distinguished from Croup. Spasm of the glottis is readily excited in infancy; irritation of the gums, or of the stomach, or an undue excitability of the nervous system, will suffice for its production without either local congestion or general febrile action. It is often first noticed as the child starts out of sleep with a stridulous or crowing inspiration checked or interrupted by the spasm; the head is thrown back and fixed, the chest motionless and the face livid; in some convulsive action a little more air may enter the chest, when in a few seconds the spasm yields sufficiently for expiration to be effected, perhaps to be succeeded by another crowing inspiration or by a more free entrance of air, and the attack terminates in a fit of crying; the breathing then regains its natural characters, no signs of stridulous inspiration remaining. An attack of this severity is seldom the first to which the patient has been subject, but it is likely to be repeated on the slightest cause of excitement or alarm; the crowing inspiration will not always be heard; there may be only momentary holding of the breath or acts of involuntary deglutition; it sometimes is induced by the act of swallowing or of suckling; or it interrupts a fit of crying, the loud and long expiratory sounds of that act being replaced by a short faint sound, and inspiration becomes long and noisy, instead of being short and free; an expression of alarm is fixed on the face, which becomes red and turgid, and the spasm may either pass rapidly into natural crying, or there may be more serious cause for alarm. The breath may be held for half a minute, the spine arched backwards and rigid, the thumbs bent inwards on to the palm of the hand, the great toes separated from the others, and both fingers and toes strongly flexed. Where the convulsive proclivity is marked, and some persistent irritation not in the windpipe keeps up cough which might mislead, this bending in of the thumbs is a valuable aid in the diagnosis. In the slighter cases the attack being accompanied by crying rather than cough, and the tone of the cry being natural, would almost suffice to distinguish these cases of child-crowing, or laryngismus stridulus, from Croup. Dr. Clarke,[1] who describes this condition accurately, remarks that "it has sometimes been called chronic Croup, but it is very different from Croup, and is altogether of a convulsive character."

Some cases of this kind have been described by Franks and Kopp, in Germany, as thymic asthma. The enlarged thymus is only one of the signs of a persistence of the infantile condition, among others a quickness of the pulse and respiration, and even the infantile ratio between these acts of 3 to 1 may be noticed: nor are these attacks limited to the earlier months of infancy, but may be continued into the fifth year; their abrupt commencement and termination, the freedom of respiration in their intervals, and a concomitant derangement of the child's health not of a febrile character, in which slighter symptoms of the same kind occur, the temperature often being considerably below the healthy standard, separate all cases of this kind from Croup.

Foreign bodies entering the glottis[2] produce croupal symptoms which are distinguished from Croup by the absolutely sudden manner in which a child, probably in perfect health at the time, is seized; it may be during a meal, or with a known object in the mouth which has disappeared while about to speak or at play, and not during sleep or at night. There is one kind of foreign body, however, which may find its way into the glottis during sleep, and that is an *ascaris lumbricoides* from the stomach; several instances of this are on record. It might seem possible that matters vomited during sleep should be drawn into the windpipe; this has happened to adults, but rarely if ever to children. The dyspnœa from any of these causes, if not suddenly fatal, is as violent at the first moment as at any subsequent time. There may be remissions from the foreign body descending into the bronchus; in this case the sound of the voice or of the cough would be clear, and there would be no tracheal siffle, while the signs of obstructed respiration, more frequently found on the right, must always point out the site of the foreign body: when it is in the larynx or trachea it gives rise to constant dyspnœa, with loud expiratory bruit as well as the stridulous inspiratory sound. The ingress of acute Croup has been attributed to a possible accident of this kind, the alarm occasioned by the sudden onset of dyspnœa having made the mother or nurse oblivious of the premonitory cough; taking the stethoscopic signs and general course of the symptoms together, the diagnosis need not long remain doubtful. When boiling water or chemically irritating fluids have been swallowed, there is no room for doubt, and the state of the mouth, pharynx, and nares, confirms the history; the whole course of the resulting lesion differs only from Croup in its sudden commencement and in its cause.

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[1] Commentaries on some of the most important Diseases of Children, by John Clarke, M.D. Part I. p. 88. London. 8vo. 1815.

[2] A case is recorded in the 1st volume of the *Med. Times and Gaz.* for 1853, p. 126, of a fruit seed, three-fifths of an inch long and one-fifth in circumference, passing the glottis of a child two years and one month old.

Injury to the larynx from without is a possibility that suggests a careful examination of the integuments of the neck for any appearance of injury, or of subcutaneous emphysema: the outline of the larynx should be traced; its mobility noted, as also whether there is marked tenseness, swelling, or tenderness, in its immediate neighbourhood; these last characters, with a limited extent of redness of the skin and loss of elasticity at the spot, might indicate deep-seated abscess.

There are other chronic affections which in their exacerbations may simulate Croup. Polypus, or growth from the mucous membrane in the interior of the larynx, may occasion croupal dyspnœa, which besides its chronic history is accompanied with stridor, both in inspiration and in expiration. Tumour causing mechanical obstruction of the trachea is rare in children. Spasm of the glottis may be occasioned by tumours, enlarged glands, or abscess, implicating the recurrent nerve; post-pharyngeal abscess may not only cause obstruction, but serious injury to the windpipe; it is generally a sequel to other diseases, and is therefore more likely to be met with in secondary Croup, of which it may not only be a complication but a cause; a digital examination of the posterior wall and sides of the pharynx would give the necessary information of its progress where inspection is unsatisfactory.

Even in acute disorders, and especially in the more urgent and sudden cases of dyspnœa, the pharyngeal surface of the glottis should be examined by touch, whereby an unsuspected cause of obstruction may be at once detected and removed. The interior of the mouth and throat should be inspected in all cases; without this the nature of the diseased action cannot be evident, and by it alone it can often be determined that some disease other than Croup is the cause of the symptoms. The physical examination of the chest must never be neglected; where Croup is present it is the chief means of tracing its progress, and, as pointed out by Dr. Williams, the first sign of the disease, even at the onset, may be detected by the stethoscope, and by the same aid some of the diseases liable to be mistaken for Croup are most easily recognised.

Catarrh in young children, with a proclivity to spasm, may occasion a hard ringing cough, attended by sibilant inspiration in the early part of two or three successive nights; the respiratory surfaces are now the source of the irritation exciting this spasmodic cough. In stronger or older children the same local affection sometimes induces a passing hoarseness or aphonia, and a short harsh cough which is husky rather than dry, and is neither frequent nor spasmodic, unless it be slightly so during the first sleep, and then even sibilant inspiration may be audible; but this again disappears, the breathing is free, there is no acceleration of the pulse, and any heat of skin soon yields to perspiration: the illness has commenced with well-marked catarrhal symptoms; the cough is at times heard with a moist sound, or is attended with secretion, and auscultation furnishes the signs of incipient bronchial catarrh; these are well developed on the second day, and become more extended; they are uninterrupted by the siffle of laryngeal obstruction, which no longer even obscures the normal breath sounds.

Hooping-cough often has the frequency, more rarely something of the tone, of the cough of Croup on its first commencement, but as it progresses the cough is attended by a shrill inspiration which differs in character from that accompanying the cough of Croup only by the sonorous quality constituting the hoop; the cough is also worse during the first half of the night, and occasions suffocative fits of dyspnœa; these are produced differently from those of Croup, and are more directly the effects of the cough, which consists of short expiratory efforts, rapidly succeeding each other, no inspiratory action intervening. Thus a turgescence and lividity of countenance is caused, which rapidly disappears after free inspiration is accomplished; it is at once obvious that sufficient air is received into the chest during the inspiration, as the sonorous quality would seem to indicate, while the perfect relief to the child, the gentle play of the chest walls, and their rounded form offer a sufficient contrast to the dyspnœa of Croup. Moreover, at the period of the illness when the hoop is developed, the cough has already existed some days with precedent catarrhal symptoms; the more persistent dyspnœa of severe hooping-cough comes on still later, but whether in its earlier symptoms or later effects, it is in the more distant air-tubes that the signs of disease are detected, and not at their commencement. The hoop is, moreover, only an occasional phenomenon, while in Croup the stridor continually increases and becomes unintermitting.

In measles a ringing cough, and dyspnœa resembling that of Croup sufficiently to mislead, sometimes accompany the catarrhal symptoms of invasion; the cough may have exactly the tone of Croup, but the stridulous inspiration is less marked, and both will sooner become catarrhal. The febrile condition is similar, so that unless the presence of measles is expected, or its signs just apparent, there may be no guide as to the nature of the affection but the character of the throat-redness; this is in patches of dusky red instead of being uniformly bright. The croupy condition induced by the ingress of measles subsides when the rash is well out; it is however liable to recurrence from various and often slight causes, for some time after the original disease has entirely disappeared.

Diphtheria implicating the air-passages produces the effects of Croup, with very similar symptoms; these, though they do not supply the main elements of the diagnosis, afford many points of difference; they are the sequel of a more general or a more prolonged diseased action, and may not appear till after three or four days, or even a week, of illness: their mode of development is most varied; sometimes they have not attracted attention until the dyspnœa is unintermitting; sometimes at their very beginning they will embarrass the respiration and excite the circulation to a degree that completely alters the features which had up to that time marked the disease; at another, in the intervals of dyspnœa, though the respiration is not free, the child will take food, resume its play, and either seem cheerful and excitable, or indifferent to its danger. Though the croupal symptoms may show an exacerbation during the first half of the night, they are as frequently first noticed at other times; they are neither so paroxysmal in their commencement, nor so dependent on the urgency of the cough. An excess of suffocative difficulty may not occur so early as in Croup, but when it has occurred there will not be so complete a remission as after the first paroxysm of Croup; the impeded respiration, with signs of laryngeal and tracheal obstruction in both expiration and inspiration, may be detected before any severe distress has been thereby occasioned. Early notice is often given by a hoarse cough, which has a muffled rather than a ringing sound, and by an unpleasant tone of the voice, which is husky rather than hoarse, or it is nasal in tone, or croaking and deep, but much sooner becomes whispering, or permanently extinct.

Where the more general characters of diphtheria are well marked, its epidemic prevalence known, a contagious influence traced, or some days of illness have preceded the attack, there will be little difficulty in rightly estimating the nature and cause of these additional symptoms, and none if the few days of previous illness have been under observation, so that the special products of diphtheria and the asthenic tendency of that disease have been recognised. Where the phenomena of dyspnœa are those first presented to our notice, it will not be possible to arrive at a diagnosis from them, unaided by inquiry as to the history of the attack; whatever the character of the dyspnœa, and however recent or even sudden the attack, if severe illness have recently been recovered from, or if there is the history of only a short illness that seemed to be passing off, it may not be diphtheria, but it is probably not idiopathic Croup; while if there have been two or more days of illness immediately preceding the first croupy symptoms, if on one day there have been dulness or debility and refusal of food, if one night's extreme restlessness unattended by cough, with excitability or irritability, headache, vomiting, an unusually free action of the bowels, or even a very free secretion of urine, coryza, and congested or glistening conjunctiva, defluxion of glairy fluid from the nostrils, or stoppage of one or both of them, creamy moist tongue, and difficult or painful deglutition, the presence of diphtheria may be inferred: there will then be greater enlargement of the lymphatic glands of the neck, and especially of those at the angle of the jaw; these may be so full as to render a free inspection of the mouth difficult, yet the enlarged tonsils, and the unequal congestion of the soft palate, will give further confirmation, even if no patch of diphtheritic deposit come into view.

The fullest and clearest inspection possible of the mouth should be obtained; one spot of diphtheritic deposit brought into view clears up all doubt in this most important matter; it may be below or behind the enlarged tonsil, or almost hidden in the angle of the soft palate and uvula, or completely concealed by the uvula or velum; it may only just be commencing in the follicles of the tonsils, or may have already cleared from their surfaces, leaving only the mark of its attachment, or a faint indication within the substance of the mucous membrane. When extensive deposit in the fauces invades the larynx by continuity, a glance is sufficient to confirm what the general aspect of the patient would suggest, and from the greater enlargement of the lymphatic glands a very limited inspection is often all that can be obtained; there would in such cases probably be considerable fœtor of the breath, and secretions escaping from the mouth without effort to expel or restrain them. But the implication of the larynx is as frequently effected by new centres of deposit as by continuity of advance, and there are not wanting cases of diphtheria to show that the diseased action may commence in the air-passages; in these cases the careful record of the mode of ingress—the attack being recent, this is more easily obtained with accuracy—and the consideration of the concurrent symptoms must be mainly depended upon, bearing in mind that it is in these cases where the excitement of the circulation and general heat of surface are most likely to mislead; the pulse, however bounding, quick, or full, is not strong or hard, and the breathing, though disturbed, is not accelerated proportionately with the pulse; indeed, during the development of diphtheria the respiration generally has a ratio of less than one to four pulsations; any precedent vomiting, diarrhœa, or diuresis, with loss of sleep, loss of appetite, or difficulty of swallowing, powerfully aid the diagnosis; while an inspection of the fauces is of the greatest importance, as some of the appearances are the same whether the first product of the disease be deposited there or not.

In diphtheria some part of the pharynx is sure to become the seat of the disease, and to show unequal redness and turgescence at some points before the deposit occurs; or if the first patch have separated from the tonsil, these signs would indicate fresh deposit elsewhere; and while it is rare for the larynx to be the part first attacked, it is very frequently invaded by extension from these new centres of deposit. The aryteno-epiglottidean folds are often the points from which such extension proceeds, and cannot at this time be brought into view; or some point near the posterior nares may be implicated, and be therefore quite out of sight; in these cases a glairy or yellowish mucus in streaks along the back of the pharynx, or collected in some quantity in the gullet, will give indications of the disease: probably other isolated patches may appear either in the nares, on the lips, or even the eyelids; the lymphatic glands are always enlarged, and the deglutition is or has been difficult. In making the discrimination between diphtheria and Croup it is necessary to detect the phenomena which are the earliest to appear, and those which are most easily ascertained, but not to neglect others which may confirm the diagnosis. The occurrence of epistaxis, or of blood in the expectoration, or of well-marked pieces of false membrane in the matters cleared from the throat with specks of blood on one surface, would point to diphtheria. The presence of albumen in the urine is conclusive in the diagnosis, and may sometimes be detected in the pale urine passed at the commencement of the attack, though frequently not appearing till a later period. Paralysis of some of the muscles of vocalization, deglutition, or of motion, is equally distinctive of diphtheria. Finally, a prolonged convalescence and a less tendency to recurrence not only confirm the diagnosis, but complete the distinctions between diphtheria and Croup.

Small-pox is sometimes attended by a consecutive affection of the larynx and trachea simulating Croup; about the seventh day of the eruption a little hoarseness, some hard cough, dry at first, but soon with expulsion of tenacious mucus, characterise a secondary croup, the result of the specific lesion of the larynx and trachea, which is rapidly fatal.

Measles gives rise to secondary Croup, only distinguishable from idiopathic Croup by its more asthenic character, and by its having been preceded by the specific disease. It does not come on till the rash is disappearing and the cough which attended the eruption is subsiding; there is frequently an aphthous condition of the mouth at the same time. We may here infer a specific action on the mucous membrane similar to that exciting pneumonia under the same conditions. Some cases of secondary Croup are subsequent to the pneumonia of measles; these[1] and many of those occurring earlier are really owing to diphtheria attacking a patient debilitated by the primary disease: diphtheria may also complicate measles.

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[1] Archives Générales de Médicine, tome vi. p. 466.

Scarlet fever may possibly excite croupal symptoms by extension of the specific inflammation of the throat to the larynx; more frequently some general turgescence of tonsils, submaxillary lymphatic glands and the parts about the throat excite croupy paroxysms. Croup secondary to scarlet fever is later in its appearance. The majority of these cases of secondary Croup are complications with diphtheria, and in the few which survive recurrence is rare.

Erysipelas is sometimes attended by croupal complication; here the dysphagia is extreme, and the epiglottis is swollen, red, and erect.

Œdema of the glottis may follow continued fever or other prolonged illness; the consequent dyspnœa comes on in the night, and may be rapidly fatal; but if a remission occur there is neither the same degree of sibilant respiration nor the cough of Croup, there is but little dysphagia, and the epiglottis is neither red nor swollen.

Inflammation of the tongue or mouth, either from the effects of mercury or other causes, may extend to the larynx. In all these cases there is the existence of the previous disease, and the affection is of the asthenic type; some of the latter are rare in children of the age at which we expect Croup.

Hysteria may come on in a subject young enough to render it important to exclude the possibility of its being Croup, so closely may it simulate the leading features of the disease. Dr. Cheyne relates a case so deceptive, that he was induced to order bleeding for its relief. This close correspondence is more liable to occur in one who has been the subject of Croup in childhood; but then, though the attacks may have been numerous up to eight or nine years of age, there has been an immunity for the past three or four years. Cough with occasionally a croupy sound is first heard, and the attack generally comes on towards night: the cough is loud and peculiar, and the respiration becomes soon noisy both with inspiration and expiration, the head is thrown back, the hands clutch at the throat, the face becomes red and swollen, the eyelids are closed and puffy, and the pupils dilated. The dyspnœa may be of some continuance, but is not really so extreme as it may seem; the auxiliary muscles of respiration are not called into service; there is no drawing in of the supraclavicular spaces, nor sinking of the epigastrium; the respiratory murmur may be weak, but is everywhere audible; there is no tracheal siffle heard by the stethoscope placed at the top of the sternum; the voice may be harsh and produced with difficulty, but is not much altered: a conclusion will be arrived at by conjoining these particulars with an observation of the general attitude, the arching backwards of the body, the tossing of the limbs, the age and sex of the patient, the absence of definite complaint, the impaired volition of hysteria, and the spasmodic muscular actions, the most obvious of which will probably be the grating of the teeth. In these cases, whatever other disordered functions may be associated with the attack, it is necessary to be satisfied that it is not owing to local irritation, and that there is no appearance of disease in the fauces.

PATHOLOGY.—Croup is essentially an inflammation of the mucous membrane at the commencement of the air-passages, and this, not of the superficial or catarrhal kind, but such as affects profoundly its texture, nutrition, and secretion. The determining cause of this, as of other more deeply-seated inflammations, it is difficult to estimate. What in one subject may excite but a passing irritation, in another sets up a definite course of diseased action, its phenomena modified by the conditions of the part in which it is developed. The vital importance of the parts which are here the seat of the disease, the special endowment of the glottis and larynx, and some peculiarity in their structure and development, determine the phenomena of Croup.

At the time of puberty the larynx rapidly increases to one-half more, or to double its previous calibre, according to sex; an increase in the capacity of the trachea has been going on for some time, but during childhood the air tube and its orifice are remarkably small; at this period the mucous tissue is capable of as much distension from turgescence of its vessels, and even of more swelling from submucous infiltration than accompany the inflammatory process in adults: hence a certain condition of the lining membrane might become a dangerous obstruction to the entrance of air in the one case, and not offer an alarming impediment in the other. The existence of submucous cellular tissue as far as to the vocal cords, conduces to this possibility. The upper part of the larynx is also finely organized for the exclusion of any foreign or irritating particles from the air-passages; its muscles are spasmodically excited directly these irritants from without come into contact with the mucous membrane covering them. In this disease, the irritant cause is from within; no sooner is the congestion of the membrane sufficient to interfere with the normal state of its surface, than a source of irritation is provided which cannot fail to induce the violent action of the muscles beneath: at first, probably, it is not so much the swelling as the dryness of the membrane, from check to its natural secretion, which is concerned in the causation of the spasm; the hoarse voice indicates turgescence of the mucous membrane as far as to the vocal cords; the tone of the cough and the intermitting laryngeal siffle show that swelling is not great, and that spasm is an integral part of the seizure. The further effects of the inflammatory action are very evident, whether its intensity be confined to its first site, or be extended further along the air-passages; in the first case inflammatory products are formed both beneath and upon the mucous membrane, in the second they are accumulated on the surface, and in either the results are commensurate with the symptoms, and afford a sufficient explanation of the resulting apnœa.

A very small amount of exudation, added to the swelling and spasm of the larynx, may not only be fatal, but produce symptoms of greater urgency than would ensue from a considerable accumulation in the trachea, and yet few traces remain after death to show that the passage of air was precluded. In the trachea all inflammatory exudation must be found upon its surface, and may there remain with less obstruction to respiration than is possible in the larynx, though not without offering a further impediment to the entrance of air into the chest; the power of the cough is thus lessened, and it is further diminished by the imperfect closure of the swollen glottis, while the secretions of the surface over which the inflammation extends are altered, so as to be less easy of detachment until the inflammatory action yields, by which time all expulsive power may be lost, so that albuminous concretions in the air tubes are a frequent post-mortem appearance, and are often most remarkable in the trachea; it does not necessarily follow that the inflammatory action has been more intense at this part, still less that this has been its starting-point.

The symptomatic fever which accompanies the disease often has its sthenic character modified at an early period by the imperfect aëration of the blood; an early indication of this is afforded by the cerebral phenomena: some of the symptoms most distressing to witness are not those most felt by the sufferer; movements at first instinctive become imperfectly co-ordinated, and with the impairment of consciousness purely convulsive; the bending backwards of the head in the latter stages is not most frequently observed when the trachea is most obstructed, but is referrible to convulsive action; and spasm may be the immediate cause of death. Whether respiration be thus suddenly stopped, or more gradually abolished, the actual termination of the disease is always by apnœa.

Morbid Anatomy.—Intense redness of the mucous membrane is persistent after death; swelling is seldom found, though sometimes the aryteno-epiglottidean folds are considerably distended, so as to diminish the upper opening of the larynx. Swelling may also be noticed at the base of the epiglottis, in the sacculus laryngis, and at the superior vocal cords and ventricles of the larynx; the mucous membrane is not much thickened, and has rarely undergone softening; sections of the mucous folds sometimes discover serum, sero-purulent fluid, or even pus beneath; pus has also been found disseminated between the muscles and cartilages of the larynx; the surface of the membrane has not lost much of its smoothness or polish; small patches of semi-transparent lymph occur, or a soft whitish exudation rests on some parts of the surface, or fills the ventricles, or in its place, viscid mucus or pus may be found on the upper edge of the glottis or filling the laryngeal pouch. Ulceration of the surface is rare, especially in acute cases; Dr. West has observed it in one case of idiopathic croup not of the most sthenic type, and more frequently in cases of secondary croup; in one such case he records[1] "an uneven granular appearance of the larynx, and ulceration of the epiglottis." In some cases of Croup secondary to measles, small aphthous ulcerations may occur above the rima glottidis coexistent with true aphthæ of the mouth; no deposit in the pharynx or slough of the tonsils is found in true Croup. Dr. Cheyne remarks[1] that "in other diseases inflammation and incrustation, extending from the fauces to the larynx, may often be observed, but in genuine Croup, as it exists in Scotland and Ireland, never." The formation of false membrane, more or less continuous, is a usual result of the inflammatory process in Croup; its most frequent situation is in the trachea, but its presence is not so invariable as to make it pathognomonic; in some cases shreds of lymph, or striæ of viscid mucus, or vivid redness only, are found in the trachea; in others a soft curdy lymph, or pus, is present in some quantity and extends into the bronchi. Where a more fibrinous exudation completely fills the trachea, it is separated from its surface by a layer of pus or of muco-purulent secretion, some of which may be found on both sides of the lower part of the false membrane, but it is less abundant and more viscid at its upper part. The false membrane may extend into the bronchi; its transition to a soft and less tenacious substance is so gradual that it may become at any point indistinguishable from the muco-pus covering the surface, which seems to be undergoing the same inflammatory process. The false membrane of Croup differs from that formed during the specific inflammation of diphtheria, both in its chemical and physiological relations; it is not simply fibrine, but consists of effused lymph in which the presence of albumen can always be chemically demonstrated: microscopically it is a mass of cystoid corpuscles; it is not the result of an interstitial change in the substance of the mucous membrane, but an exudation from its vessels and glands, so that the structure of the membrane producing it remains singularly free from pathological injury. This point has attracted the attention of many of the observers of Croup. Albers, of Bremen, records the absence of all traces of inflammation of the subjacent mucous membrane in some of his cases, and considers that the plastic exudation being accomplished, the inflammation not only diminishes but probably ceases altogether. He notices also cases of another kind, and it is probable that two classes of disease were before him. MM. Barthez and Rilliet,[2] who represent the French school, in applying the terms Croup and pseudo-membranous laryngitis only to cases of diphtheria, remark "that the mucous membrane beneath the false membrane presents very various conditions,—it is sometimes perfectly healthy; this is a fact we have ourselves established." And again, alluding to the number of cases of severe Croup on record where mere shreds of false membrane were found in the larynx and trachea, an instance of which they themselves observed, they ask, "May there not yet be a disease which presents so great a similitude to Croup that it has been confounded with it by most authors, and in which there is found after death either no alteration in the mucous membrane of the larynx, or a simple inflammation, without swelling enough to obliterate the air-passages?"

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[1] Lectures on the Diseases of Infancy and Childhood, by Charles West, M.D. 2d Edition. 8vo. Lond. 1852. P. 220, note.

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[1] Cyclopædia of Practical Medicine. Article, Croup, by J. Cheyne, M.D. London, 1833. Vol. i. p. 499.

[2] Traité des Maladies des Enfants. Paris. 8vo. 1843. Vol. i. pp. 320 and 336.

It is such a disease that we mean by Croup; and while in young children simple turgescence of the mucous membrane conjoined with spasm may be too soon fatal for the special products of inflammation to be found, and in others these products collected near the base of the arytenoid cartilages and in the sacculus laryngis may occlude the glottis at an early period of the disease; yet a more or less continuous membranous exudation may in other cases completely fill the trachea, and even extend into the bronchi; whether it is ever so closely identified with the mucous structure, or so purely fibrinous as is the special product of diphtheria, will require further observations to decide. In some cases the false membrane rapidly forms again after expulsion. Sir Thos. Watson relates a case[1] where after tracheotomy it was completely re-formed in six or seven hours: the rapidity of its formation indicates a developed speciality rather than an original intensity of inflammation, and numerous instances prove that the most vigorous antiphlogistic treatment has no effect in retarding its development, while in some acutely inflammatory cases of Croup going on to their fatal termination, no false membrane has been expectorated, and pus only has been found in the windpipe.

The bronchi show more or less trace of inflammation in their whole extent: in the upper tubes a whitish concrete exudation is often developed, and sometimes presents sufficient tenacity to be removed entire from one or two divisions; the smaller ramifications are often filled with opaque mucus but slightly aërated. Pneumonia both lobar and lobular frequently coexists, and vesicular emphysema is generally present in some part of the lung. The heart may contain dark blood, both in its right cavities and in the left auricle, and the venous trunks and sinuses are similarly distended; there may be congestion, but there is no fibrinous deposit found in the liver, spleen, and kidneys. The brain may show congestion of its vessels or slight serous effusions within its ventricles. Numerous enlarged lymphatic glands are found beneath the thyroid on each side of the trachea; sometimes lymph has been effused both in this situation and between the sterno-hyoid and sterno-thyroid muscles.

PROGNOSIS.—We have no data from which to judge the proportion the mortality from Croup bears to the number of the attacks. The fully formed disease is one of the most fatal to which childhood is liable, and the younger the sufferer the less favourable the prognosis. Were every case considered as one of Croup, in which hoarseness of voice and peculiarity of cough lead to the prompt and careful adoption of the means necessary to ward off the disease, we might under the most favourable hygienic conditions come near to the hopeful conjecture of Dr. Wood, of Philadelphia, that one case in fifty only is fatal. The mortality estimated by Jurine, of one in ten, is probably nearer to that we experience; where it has been placed as high as one-half, two-thirds, or four-fifths, cases of diphtheria are included; nor is it surprising that the most fatal complication of a very fatal disease should be thus mortal when, under a misconception of its nature, the course of treatment pursued has been such as would endanger the result of uncomplicated diphtheria. The inherent fatality of secondary Croup is also increased by subjecting it to the course of treatment required in uncomplicated cases, and the favourable prospect of some of these is sometimes in jeopardy from the too prolonged use of tartar emetic. The slightest cases of Croup furnish grave cause for anxiety; for some of the most severe there is a hope in reserve, faint though it be, which is offered in few other acute diseases. Early treatment has a great effect on the favourable result of even the most severe cases. The most active treatment will often be of the least avail in the advanced stages.

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[1] Lectures on the Principles and Practice of Physic. 4th Edition. Vol. i. p. 856.

The signs of a favourable progress are a softening in the tone of the cough, a diminution in the frequency and severity of the attacks, with a more free entrance of air into the chest, a less distress of breathing in the intervals, a return of the voice towards its natural tone, a loose or moist sound with the cough, and the possibility of a comfortable repose. This change in the symptoms must be continuous for twenty-four hours, and no lung complication present itself, before confidence is warrantable.

If the paroxysms be more frequent and more violent, and there be no diminution of the dyspnœa, or an increased restlessness in the intervals; if the voice be suppressed, and the cough less powerful or absent; if the expansion of the lung be reduced till the chest walls seem retracted or flattened, there is no hope of a favourable change taking place; and even if death be not sudden, it will come on more slowly, but not the less surely, with evidences of disordered consciousness and coma: where this stage is reached, though from some unexpected relief air has been again admitted into the lung, and some of the functions of life are resumed, and even continue for a time, recovery is scarcely possible.

TREATMENT.—Few diseases are so dependent for their relief on the prompt and careful adaptation of a thoroughly antiphlogistic treatment at their very commencement as is true idiopathic Croup. Unless the first threatening symptoms have received all the attention they demand, a free abstraction of blood may be indispensable in checking its progress. The induction of vomiting in the early stages is often the most effective means of arrest, and one that must be resorted to in all stages of the disease but the last. The air to be respired will require its temperature elevating and made equable by day and night, and aqueous vapour should be diffused therein. The warmth of the surface and the application of warmth externally must be attended to, and diluents only allowed to be taken.

The exclusion of every possible irritant, as well as moderating the degree of irritation already existing, are of the utmost importance, either in preventing the progress of the threatened malady, or in favouring its arrest and subsidence.

However urgent the symptoms, attention must first be directed to their course and development, and their cause ascertained; before our special remedies are employed steps can be taken to secure those general aids which are indispensable. There must be a fire in the room, and a large quantity of hot water in readiness for a bath must be prepared; meanwhile some hot water can be brought to the bedside, sufficient perhaps for the immersion of the child's arms, the steam from it may be of some service, or sponges wrung thoroughly dry from it and repeatedly applied to the throat will afford some relief; the nurse or chief attendant upon the child must be retained in the room not only to assist in these measures, but to afford the necessary information as to the commencement of the attack and its antecedents, while at the same time the degree of embarrassment of the breathing, the state of the pulse, of the cervical glands, and of the surface generally, can be noted. An emetic should be in readiness, either the ipecacuanha wine, or the antimonial wine, or both; a combination of the two in equal proportions is well suited for the earliest stages, or a mixture of twelve grains of powdered ipecacuanha with a drachm of syrup, or of mucilage, and three drachms of water, will be of the same strength as the wine; an aqueous solution of the tartarized antimony of any strength can be made by means of hot water; some of this solution, containing at least a grain of antimony, must be added to the above mixture, the half of it given early to excite vomiting, and the remainder in less than half an hour, if that action has not commenced. In this interval an inspection of the throat should be obtained, and care must be taken to make it thoroughly efficient; a good light is required; if artificial light is needed, three candles cemented together will afford it; the child must be well secured in the nurse's lap, the head resting against her shoulder—sudden movements of the head, arms, or feet, must be guarded against, and by passing a firm unyielding instrument, or the broad handle of a tablespoon, to the back of the tongue, it can be depressed and at the same time brought forward so as to show the epiglottis. This inspection may be convenient before the first emetic dose is given, and if any doubt exist as to the nature of the attack, would then be advisable; whenever it is accomplished the diagnosis is complete, and the activity of the means employed can be proportioned to the severity of the disease, the stage at which it has arrived, and the condition of the patient.

Before repeating the emetic it will be advantageous to make use of the warm bath: this should be of a temperature of 98° or 99°, and maintained at the higher degree of heat, or even raised two or three degrees above it by the gradual addition of hot water; a warm blanket must be in readiness to envelope the child on removal, and hot towels to dry the surface completely. The air of the chamber should be raised to a temperature somewhat over 65°, and never allowed to fall lower; it can be made moist by placing a kettle of boiling water on the fire with a tin tube fixed to the spout, or a long roll of paper, to convey and diffuse the steam. After the action of the emetic there will generally be great relief to the distress of breathing, and quiet sleep will follow. It is now the time to listen attentively to the breathing; however complete the relief afforded by the emetic or the warm bath may be, the patient must be seen during sleep, or visited again at night, not only to see that the improvement continues, but for the purpose of noting the state of the respiration; the stethoscope should be applied to the side of the neck or to the top of the sternum to ascertain the character of the tracheal bruit, to the upper part of the chest in front and to the lower part of the side or back, in aid of other observations as to the degrees of freedom with which the air is entering. If the result be satisfactory, it may be sufficient to prescribe ten grains of alkaline citrate of potash, or five grains of chlorate of potash, to be taken every four hours, freely diluted with water or with milk and water, and a grain or two of calomel for its aperient action, which may be aided if necessary by castor oil in the morning; it is better to produce this effect by giving three or five grains of calomel at once, should the child have gone through a severe paroxysm of dyspnœa. If there be persistence of the febrile symptoms, of the cough, or of any laryngeal quality in the breathing, half-drachm doses of antimonial wine must be given with each dose of the saline, or a smaller dose at more frequent intervals, taking care with young children that it is given less frequently as the symptoms subside, and that it is omitted altogether as soon as relief is obtained. The emetic must always be at hand, so that in cases of a threatened paroxysm its full effect may be again induced. It is to be borne in mind that the paroxysm has a tendency to recur even when the disease is not advancing, and that the recourse to the warm bath may afford such relief as to enable the air to be drawn in again either with freedom or with lessening signs of obstruction.

The attack, when treated early, is not unfrequently arrested; the patient requires to be carefully watched, that any return of the croupy symptoms may receive timely attention; however favourable the progress, the child should be confined to bed for two or three days, the diet being gradually increased; the temperature of the room is to be maintained during this time, and great caution exercised before allowing the patient to leave it, the chest symptoms meanwhile being anxiously observed. Where the season is cold, or unusual susceptibility has been induced by repeated attacks of Croup, it may be necessary to restrict the patient for ten days or a fortnight to an apartment wherein the air is artifically moist and warm.

In the more severe cases a tent should be formed over the child's bed, to which steam can be admitted from the long spout of the kettle, which, filled with boiling water and placed behind the bed, can be kept hot by a spirit lamp or other means; the temperature within can be regulated from 70° to 75°, but at times it may be raised to 80°, and a larger quantity of steam be admitted with advantage. Calomel should be given from the first, and repeated frequently in small doses, interrupted occasionally for the repetition of the emetic; a grain, or half a grain of calomel, combined with the quarter or eighth of a grain of ipecacuanha, according to the age of the patient, is to be prescribed every two hours; if the bowels become disturbed, it can be persisted with in diminished doses until its characteristic effect on their secretions is obvious, nor is it then to be entirely discontinued. Antimony has great power in moderating the intensity of the first stage of the inflammation; it may, therefore, be used alone to cause vomiting, and as it will be necessary to repeat it with this object, it is better not to give it in frequent small doses, which, by inducing in the system a tolerance of its influence, render its emetic effect less easily obtained; for the same reason it is not to be the only emetic employed; where it is of service its good effect is soon observable, and can be secured by giving it in small doses with other emetics. In the later stages of the disease it is entirely inadmissible.

In some cases, the necessity for bloodletting has to be considered in the earliest stages of the treatment: in certain districts, where the subjects of the attack are well nourished, and living much in the open air, the early abstraction of blood has been found by experience to be a main element in the favourable issue of the illness; there are also certain cases where the attack is of such severity that the influence of the emetic is better aided by the loss of blood than by the use of the warm bath. In these cases the bleeding should be practised before the action of the emetic has commenced, and the blood should be withdrawn rapidly, so that the system may at once feel the effect of the loss; the external jugular vein affords the requisite flow most readily; it may be obtained from the arm, except in young children, and with them leeching is not so efficient a substitute as might be expected. The youngest children bear the loss of one or two ounces of blood better than abstinence from food, or the effects of depressing medicine; for a child of four or five years of age, bleeding to the extent of three or four ounces will suffice, either for present relief or for a check upon the advance of the disease; it is to be remembered that in the cases where it is most indicated, neither of these objects will be attained by this remedy alone; even where the immediate relief is great, in a few hours there will be a return of most of the symptoms which generally indicate its employment, and these will require for their control many of the means already mentioned, probably with the aid of local bleeding. General bleeding is only of service in the early stage of the disease, it is not to be repeated; loss of blood is only allowable while there is heat of skin, florid hue of face and lips, and firmness, as well as fulness of pulse, but these conditions do not of themselves demand it; it is to be sparingly resorted to among town populations; it is seldom advisable where the attack accompanies any marked deterioration of health; and it is contra-indicated in almost all cases of secondary Croup.

Local bleeding by means of leeches is of great service wherever the disease is progressing towards its full development; the relief thus obtained is often very great, and may be afforded more than once if other considerations do not render such means of relief unadvisable; the influence of even a moderate loss of blood in this manner may either favour the subsidence of the disease, or the specific action of calomel upon its products, while in conjunction with the warm bath it may often replace with advantage the too frequent repetition of antimony. Leeches are best applied over the mastoid processes, or a little lower on the neck if a larger number are to be used or a free afterflow from their bites is desirable; in the former situation these can be readily closed by means of dry lint aided by pressure if required, in the latter they can be covered with a linseed poultice.

External warmth to the neck and chest is useful; it can be applied without the necessity of frequently disturbing the child, by cloths wrung out of hot water and covered with warm towels or by oil-silk and handkerchiefs, or by small bags containing heated bran, which can be accommodated to the child's changes of position. Care should be taken that no part of the surface is chilled, and dry, warm flannels should be from time to time applied to the body, legs, and feet.

Counter-irritation is of doubtful efficacy; the application of tincture of iodine to the sides of the neck is of some service, and acts more beneficially when it is covered with water-dressing. Linseed poultices, not too moist, with which a little mustard is mingled, may be usefully applied to the back of the neck and shoulders, or even to the legs: where signs of bronchial irritation are found at any part of the chest, or there is less expansion at one part than another, the advantage derived from having that part covered with a large simple linseed poultice is very great; if the other symptoms are favourable, it may be sufficient to use stimulating friction over these parts three times a day when the poultice is changed. A blister may be required for more serious pulmonary implication, but must be strictly limited to a particular spot, and should be so dressed with cotton-wool as to give rise to no ulterior pain or discomfort.

Opiates are to be avoided; sleep is needful, and will naturally happen while the disease is within safe limits, then the easy respiratory movement and increasing roundness of the chest present a strong contrast to the flattening and retraction observable during restlessness and excitement; sleep will continue while that normal condition of the chest is maintained, if not, it is soon interrupted, and there would be danger in its being artificially prolonged.

The strength is to be carefully guarded during the necessary contest with disease, and requires early support; milk and farinaceous food, as well as whey and barley-water, are soon necessary; if the attacks of dyspnœa have been severe, beef-tea or chicken broth may be given early; if distasteful, or tending to excite spasm in deglutition, they are to be given in small quantities by the rectum. The various meat essences, prepared after Liebig's formula, are very serviceable, as they contain the restorative salts of the flesh without any of the protein compounds requiring gastric digestion.

Alcoholic stimulants are injurious, until the primary obstruction to respiration is overcome, and some pulmonary complication is the source of danger. The stimulant expectorants are often required, and when repeated emetics are indicated in the decline of the disease a stimulant should be combined; ammonia, or the ammoniated tincture of valerian, may be added to the wine of ipecacuanha given for this purpose; the tincture of lobelia may also be cautiously used as an adjunct, but not if there be much obstructing secretions; senega is here of the greatest value, either in large, repeated doses as an emetic, or in combination with ammonia and squills as an expectorant; for this purpose, when the occasion arises, a grain of carbonate of ammonia with two or three minims of tincture of squills and a dessert-spoonful of infusion of senega may be given every two or three hours, as prescribed by Dr. West, mixed with a very little milk and sweetened with treacle or coarse sugar; the infusion of senega should be made with an ounce to ten ounces of boiling water, or of double the pharmacopœal strength, and its pungency shielded as above, or by the addition of glycerine; it may be freely given for its emetic effect, after the first urgency of the attack has subsided, and it may be aided in its action by the addition of ipecacuanha wine and of tincture of squills. When emetics are most beneficial, care must be taken so to regulate their employment as not to interfere with the necessary absorption of nourishment from the stomach, as sustaining the integrity of the vital powers is an important element in calculating the hopes of a final recovery.

From a very early period in the treatment of Croup, from the earliest if the disease have made some progress before treatment has commenced, the necessity for affording relief by the operation of tracheotomy has to be carefully considered, and steadily kept in view; at any moment the best judged means of treatment may be rendered nugatory by threatened suffocation, and whenever this is imminent tracheotomy is to be performed. However insidiously the condition of apnœa may come on, if it be advancing and its course unvaried by temporary improvement, unless we are satisfied that the cause of apnœa is in the pulmonary tissue itself, and not chiefly in the primary air-passages, no period of the disease, nor the surrounding conditions of the patient, scarcely even the age of the sufferer, should determine us to withhold this chance of life. The extension of the disease beyond the point at which the trachea is to be opened does not prevent the success of the operation; actual inflammation or consolidation of the lung, which would preclude recovery, will have been ascertained in its course, and can be determined by physical examination of the chest at any period; any extension short of this cannot be so determined when the distress of breathing is at its height. Where a certain extension seems probable, the operation is not always contra-indicated; the admission of air restores vitality to the system, and affords a mechanical aid in expelling the morbid products, thus tending to prevent further change in the lung itself. The surrounding conditions that are essential in undertaking tracheotomy are the same that are necessary for the successful treatment of cases where this extreme means of relief is not demanded; some approach towards securing these can be made, whatever the social state of the patient. The treatment is not to be discontinued after the urgent signs of distress are obviated by tracheotomy; and though there be now no longer need for some of the medicines, greater attention is, if possible, required in regulating the state of the air to be respired. The most frequent cause of death after the operation, next to the use of too small a tube, and the risk of its becoming obstructed by secretions, is extension of the disease to the lung; this is the natural termination of the disease, whenever the state of the trachea allows it to run its course.

The age of the patient has hitherto been closely connected with the success of tracheotomy; the unfavourable result of this operation among young children has seemed to be from the difficulty at this period of life of counteracting the asthenic tendency of the disease for which it has been chiefly practised; some results of this operation at different ages are given by M. André,[1] when Interne at the Hôpital des Enfants Malades, from which it appears that while of the cases beyond six years the recoveries are one-half, they are not one-fourth of those under that age, and of six cases under two years old there was not one instance of recovery. Most of the statistics relating to tracheotomy are drawn from cases of diphtheria, so that analogical deduction from them is unsafe; they show, however, one uniform result that is doubtless applicable to the disease and to this climate, that the relative proportion of cures increases with the frequency with which the operation is attempted. Dr. Buchanan of Glasgow has operated[2] twenty-six times with nine recoveries; of eleven cases operated upon by Dr. Cruickshank during two years in a wild country district in Scotland, eight were successful.[3] Mr. Spence[4] of Edinburgh has published some most interesting and instructive reports of cases of Croup, in eight of which he performed tracheotomy with three recoveries. The facts brought together by Dr. Fuller[5] in his valuable paper on tracheotomy in Croup, and the improvement in the tracheal tube, recommended by him and M. Gendron de l'Eure, contribute powerfully to this conclusion. The want of success with children under three years of age in this country ought not to discourage the operation. Mr. Henry Smith[1] details two instances, one a child of eleven months, and the other two years, wherein the difficulty of maintaining free the opening into the trachea seemed the only obstacle to recovery. It is somewhat remarkable that in the first three successful operations in this country for this disease no tracheal tube was used: two of these cases are recorded in the 3d and 6th vol. of the Medico-Chirurgical Transactions; the third was by Mr. Carmichael of Dublin, in 1820,[2] who maintained the opening for a week by the aid of tin retractors acting on the edges of the external wound. One of Dr. Buchanan's most urgent cases was brought to a successful termination without the use of a tube.[3] Dr. Wood, in his treatise on the Practice of Medicine, 5th edition, vol. i. p. 865, mentions three cases in which Dr. Pancoast, of Philadelphia, "removed a small piece of the trachea itself, thereby superseding the necessity of the canula, and avoiding irritation from that source;" two of these were successful. The fear of subsequent narrowing of the trachea renders this objectionable; passing a ligature through each edge of the tracheal wound might keep it open with safety for a short time; or a piece of wire bent like an eye-speculum, as made for me by Coxeter, might be used to separate the edges. No tube with less than a quarter of an inch diameter is sufficient to carry on respiration; at a year old such a tube cannot be introduced into the trachea; it would not be tolerated at two years old, so that at these ages other means must be looked for to secure a passage for the air: if it should prove that in a certain class of cases the mere opening of the trachea is sufficient, and that recovery is frequently possible without the introduction of the tube, then there would be room to hope for success even in the youngest children.

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[1] On Tracheotomy in Croup, by M. André, Bulletin de Thérapeutique. Paris, 1857. Tome ii. p. 471.

[2] Tracheotomy in Croup and Diphtheria (additional cases), by George Buchanan, A.M. M.D. Glasgow, 1866.

[3] The Science and Practice of Medicine, by W. Aitken, M.D. Vol. i. p. 587. (Third Edition.) London, 1864.

[4] Edinburgh Medical Journal, 1860, p. 693.

[5] Medico-Chirurgical Transactions, vol. xi. p. 50. London, 1857.

One great cause of non-success from this operation has been owing to cases of diphtheria being mistaken for Croup. The striking difference in the character of the two diseases, and in the treatment they require, struck me forcibly during the observation of some cases at the Hôpital des Enfants Malades, under the care of Trousseau, in the summer of 1850; the want of success in the earlier operations at that institution may partly be attributed to such cases being treated as Croup in our sense of the word. The favourable result in some cases recorded by Dr. Conway Evans[4] may be attributed to his recognition of their true relation in this respect. During the period 1850–53, but few of the croupal affections that came under my notice at the Marylebone Infirmary were cases of diphtheria; during the last ten years the majority of cases in London requiring tracheotomy have been cases of this kind.

For the operation a good light is indispensable; also, two assistants, one of whom may be the nurse, but two in addition are preferable;

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[1] Medical Times and Gazette, 1853, vol. i. p. 244.

[2] Transactions of the King and Queen's College of Physicians in Ireland.

[3] Glasgow Medical Journal, January 1st, 1862.

[4] Edinburgh Medical Journal, 1860. Vol. v. p. 400.

an unyielding cushion to support the shoulders, so that the head may rest well back upon the table beneath. An incision not less than an inch and a half in length is to be made exactly in the middle line; three ink-spots on the skin may be made to indicate this, and further, to avoid any superficial vein, the skin should be pinched up on both sides, and transfixed with the knife cutting outwards. Each layer of fascia is to be divided on a director, the knife-edge always being turned from the sternum, the isthmus of the thyroid is to be pulled upwards by a blunt hook, and the cellular tissue at its inferior border parted by the director, with as little use of the knife as possible; nevertheless the trachea should be bared over the line at which it is to be opened; the edges of the external wound are to be held apart by blunt hooks or wire retractors; small sponges affixed to stems are useful; all hæmorrhage is to be restrained before the trachea is opened; a sharp hook may be used to fix the trachea, or it may be seized below with the artery-forceps. I have seen no sharp-pointed director suited for perforating the trachea and guiding the knife securely; having the thyroid drawn well up and shortening the knife in the hand with the edge directed upwards, suffices to enter the trachea safely and to secure a well-placed opening. The expanding forceps or dilator is a useful aid in the introduction of the tube. However retracted the chest-walls, some escape of air and mucus generally follows the knife, and then a full inspiratory action restores at once the normal outline of the chest; if respiration have ceased, it would be desirable by means of a tube to make suction from the trachea before commencing artificial inflation of the lungs or the auxiliary respiratory movements. Chloroform has been used safely in this operation on several occasions, but it may be better in some cases to abstain from any addition to already existing causes of apnœa, although it much facilitates the operation. The tube introduced should always be the spring-sided, bivalve one; the inner tube should have an opening in its upper convex surface, as used by Liston; after a time a valve fitted to one of this kind, as recommended by Mr. Thomas Smith,[1] to admit of inspiration through it, and of expiration through the larynx, is a valuable addition; the collar recommended by M. Trousseau advantageously intervenes between the outer extremity of the tube and the skin.

After the operation free use is to be made of nutrient and remedial enemata; liquids can sometimes be swallowed with ease, at others they require always to be given in the form of sop.

Secondary Croup requires support or stimulation from the first; no emetics more depressing than ipecacuanha wine are available for repetition; this one answers very well when they are only required to meet the nocturnal remissions, at other times this interferes too much with the desire for nourishment; small doses of sulphate of copper in solution, as proposed by Hoffman, act well as an emetic on repetition; in some cases alum, as recommended by Dr. Meigs of Philadelphia, teaspoonful doses of the powder being given in honey or syrup every ten or fifteen minutes; it is rarely necessary to give the second dose, and the emetic effect may be obtained several times a day without exhausting the patient. An occasional dose of calomel is of service; there is no necessity for the continuous use of mercury, and its full influence is prejudicial. Great benefit often results from the administration of full doses of perchloride of iron. Salines are only admissible when Croup complicates the early stage of measles, at other times and in the other exanthemata the mineral acids are better adjuncts. The air to be respired need not be kept as warm and moist as in primary Croup, but there must be the same care as to its purity. Close attention is to be given to the state of the fauces; local applications are of service here both as a stimulus to the mucous membrane near the entrance of the larynx, and for the removal of mucosities which offer some impediment both to respiration and deglutition. If nourishment and wine are not readily swallowed, nutrition is to be supplemented by the injection per rectum of small quantities of beef-tea, to which a little brandy must occasionally be added.

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[1] The Obstacles to the Re-establishment of Natural Respiration after the Performance of Tracheotomy, by Thomas Smith, F.R.C.S. A paper read before the Medico-Chirurgical Society, June 27th, 1865.

Varieties.—There is great difficulty in defining the varieties of Croup that have been described: the greater number of them depend either on the inclusion of other diseases, or on a misapprehension of the nature of this; the terms spasmodic and nervous have been applied to the most acute inflammation of the trachea, as well as to spasm of the glottis; while pseudo-membranous, inflammatory, or asthenic have probably included more cases of diphtheria than of Croup, Catarrhal Croup has been applied to all these varieties when they fortunately have had recovery as their one common result. Croupal catarrh may exist independently, the inflammation being superficial and, under favourable circumstances, rapidly passing off: stridulous laryngitis, as used by M. Bretonneau, may be taken as its type; though not requiring the most energetic treatment, it must never be neglected. The temperature of the patient here affords the most valuable indication of the gravity of the disease. In merely spasmodic attacks there is little or no elevation of temperature. The stridulous laryngitis or false Croup of M. Guersant includes many of the slighter cases of diphtheria as well as of Croup; he has remarked[1] the frequency with which it occurs among the upper classes of Paris rather than among the poor, and that it is sometimes observed in connexion with exudation on the fauces,[2] a complication which he justly considers as "fort embarrassante pour le diagnostic;" both these circumstances prevent the terms False Croup and Catarrhal Croup from being considered equivalent. Epidemic Croup is strictly diphtheria: when that disease prevailed epidemically in England at the end of the last century, any fresh outbreak of it was so spoken of; an outbreak at Chesham, in Buckinghamshire, in 1793, carefully described by Mr. Rumsey, leaves no doubt on this point; sometimes on its appearance in a fresh locality it was simply called Croup, and the word excited as much terror then, as diphtheria has again given us reason to associate with the disease it now designates.

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[1] Archives Générales de Médecine, tome xvii. Croup et Pseudo-Croup. M. Blache, p. 493.

[2] Ibid. p. 507.

DIPHTHERIA.

By William Squire, L.R.C.P. Lond.

Definition.—An acute specific disease, both epidemic and contagious, characterised by a special inflammation of the mucous membrane, chiefly of the pharynx and first air-passages, attended with enlargement of the lymphatic glands, a rapid exudation either of fibrine or non-organizable lymph, and its deposit within and upon the surfaces affected. Other parts of the mucous membrane and the skin sometimes suffer at the same time, and changes often take place simultaneously in the spleen or kidneys, albuminuria frequently occurring at an early period. The disease is accompanied by great prostration of the vital powers, and is followed by a remarkable series of local lesions of innervation; the tendency to death is by asthenia, either coincident with the disease or gradually induced, or by apnœa from implication of the air-passages, which may happen as early as the second day, or as late as the second week, of the disease.

Synonyms.—Cynanche; Angina, Celsus; Synanche, Aëtius Aurelianus; Ulcera Egyptica vel Syriaca, Aretæus; Crustosa et Pestilentia Tonsillarum Ulcera, Aëtius Amidenus; Ulcera Pestifera in Tonsillis, Paulus Ægineta; Morbus Suffocans, Villa Real; Angina Exulcerata Maligna, Nuñes; Faucium Ulcera Anginosa, Mercatus; Angina Maligna, Heredia; Carbunculus Faucium Anginosus, Riolan; Morbus Strangulatorius, Aëtius Cletus; Morbus Gulæ; Aphthæ Malignæ; Angina Puerorum Epidemica, Bartholin; Prunella Alba, R. James; The Sore-Throat attended with Ulcers, Fothergill; Malignant Ulcerous Sore-Throat, Huxham; Angina Infantum, Wilcke; Angina Polyposa sive Membranacea, Michaelis; Cynanche Pharyngea Epidemica, and Epidemic Croup, Rosen; Angina Suffocativa, or Sore-Throat Distemper, Bard; Mal de Gorge Gangréneux, Chomel; Angine Couënneuse Pharyngienne, and Croup in the Adult, Louis; Pellicular Angina, Diphtheritic Angina, Diphthérite, and Diphthérie, Bretonneau; Pharyngite Pseudo-membraneuse, Rilliet and Barthez; Cynanche Membranacea; Cynanche Maligna, Putrid Sore-Throat, Malignant Quinsy, England; Garottillo, Spain; Strypsiucka, Sweden; Rachen-Croup, Germany.

NAME.—Diphtheria is derived from διφθέρα, a skin, or covering of leather. The multiplicity of synonyms has been occasioned by the undetermined nature of the disease, and its appearance at times and places removed from each other, either by considerable intervals or by imperfect communication. Fothergill has the merit of setting forth, during its prevalence in the last century, the identity of the epidemic with that of the preceding century; while the Spanish and Italian physicians had already recognised its correspondence with the accounts derived from antiquity. Diphthérite, as signifying the special product of a specific disease, was proposed by Bretonneau. To him we owe not only the name but that energetic inquiry into the disease, as it showed itself in France during the present century, which has led to a more perfect definition of its character, and a better comprehension of its relation, than was previously possible. He at first applied the term to the whole disease, as well as to its characteristic morbid product, so soon visible in the fauces of those attacked; but further observation, proving that the local appearance was only one of the manifestations of the more important general affection, induced him, while retaining diphthérite in its more restricted sense, to speak of the general disease itself as diphthérie. We are ndebted to Dr. W. Farr for the introduction of Diphtheria into our nomenclature as soon as the epidemic spread of the disease among us rendered it important that it should be designated by one general term.

HISTORY.—There is little doubt that Diphtheria, like the other acute specific diseases, has existed as long as the history of man extends. We have traces of it two thousand years ago, and the description given of it more than a thousand years since applies equally to its appearance in our own day. Its individuality is not difficult to recognise during its epidemic prevalence, at other times ts distinctive characters have been merged with those of scarlet fever and erysipelas; these two diseases, though specifically different, approach at many points the nearest to it: they were not discriminated even in Sydenham's time; and though scarlet fever then began to be eparated, and to receive an increasing share of attention, the intimate onnexion always observed between it and Diphtheria precludes our astonishment at finding them sometimes confounded.

Unmistakeable evidence of the existence of this special form of disease is found wherever medical science has attained any degree of xactness. Hippocrates describes it, and gives us the name of probably its first recorded victim.[1] It attracted the attention of Asclepiades and Celsus.[2] Aretæus[3] is the founder both of our knowledge f, and treatment of the disease; and Aëtius of Amida showed an qually familiar acquaintance with it. It is impossible to say which f the many plagues of the dark ages of history may claim this disease as its agent. After the time of Paulus Ægineta only the writings of the Arabian physicians can be appealed to, and it is not until the intellectual revival of the sixteenth century that we again find its traces recorded;—as by Forrestus, in Holland, in 1557; Weir, in Basle, in 1567; by Baillou, in Paris, in 1576; and by Spanish writers from 1581 to the close of the century, whence is dated the first clear account of its epidemic prevalence in modern times. The numerous and graphic accounts of many and able Spanish medical writers of the seventeenth century afford valuable materials for the comparative study of the disease; and though the Sicilian and Italian writings of the same time are less original, we gather from them many important particulars. The epidemic attracted attention in the kingdom of Naples in the year 1618, and is described by Sgambatus, Carnevale, and Nola. The writings of Cortesius, in 1625, and of Alaymus, in 1632, speak of its ravages in Sicily; at the same time it had extended into Central Italy, as witnessed by Aëtius Cletus; Severinus and Bartoline speak of its continuance. The disease, if not epidemic, continued to be extremely prevalent in Spain; and, besides the special works of Fonteccha, Villa Real, Herera, Tamayo, and Nuñes, was treated of systematically by the royal physicians, Mercatus and Heredia. Scattered notices of its occurrence elsewhere appear in subsequent publications, but it is not until near the middle of the next century that we find it extensively epidemic, appearing simultaneously in Italy, France, and England, as evidenced by the independent observations of Ghizi, in Cremona, 1747; Arnault, in Orleans, 1748; and Starr,[1] in Cornwall, 1749: it also called forth the noble essay of Fothergill,[2] published in 1748; the epidemic had then hardly attained its height in England, though cases had been observed as early as 1739. It existed at Rouen at the same time, and appeared more extensively in 1748,[3] when the epidemic was at its height in France. Sweden suffered from it in 1755, and occasional outbreaks occurred there till 1778, as we learn from Michaelis. The presence of the disease in North America at this time is described by Dr. Samuel Bard, who having been trained in the University of Edinburgh to the study of medicine, returned to its practice in his native country, and furnished, by his careful observation of the cases under his care, the foundation upon which our knowledge of the disease was to be much further advanced.

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[1] Hippoc. Epid. lib. v. tex. 37.

[2] Celsus, lib. iv. cap. 4.

[3] Aretæus, De Causis et Signis Acutorum Morborum, lib. i. cap. 9.

That each of these epidemics preserved the essential characteristics of the disease as now observed, two quotations will suffice to show. Of the first, in Italy, Aëtius Cletus, in the introductory chapter to his work,[4] the only part of interest, says, "Morbi facies hæc est. In faucibus rubor apparere incipit, cum dolore, et febre; paulo post præbet se conspiciendam pustula, quam subsequitur cum crusta ulcus cinerei coloris, quod frequentius accidit, subalbicantis nonnunquam, vel nigrantis coloris. Sæpius sine pustula crustosum ulcus; . . . . et in ipsis faucibus sunt, quibus ex ulcere itur in gangrenam, et sphacelum, qui ex œsophago porrigitur ad ventriculum, vel ex aspera arteria ad pulmonem; et hi difficulter respirant, et non nisi recta cervice, illi deglutire nequeunt . . . . ex his nonnulli profundiori somno oppressi moriuntur; alii copiosa cum narium hæmorrhagia vita finiunt, alii absque his symptomatibus exanimantur. Morbus impuberes precipue invadit, tanta cũstrage, ut familiarum multarum omnes emori visi sint. Nulla moriendi est certa dies . . . . die quarto decimo elapso non absque delicto quis emoritur—ex iis, qui pristinam valetudinem consequuntur anni plus minusve spatio, omnes fere mussitant, et verba difficulter efferunt." Of the next, Dr. Starr[1] writes in 1749: "The morbus strangulatorius, with great propriety and justice thus denominated, has within a few years raged in several parts of Cornwall with great severity. Many parishes have felt its cruelty, and whole families of children, whence its contagious nature is but too evident, have, by its successive attacks, been swept off. Few, very few, have escaped." "Many in the first attacks have complained of swelling of the glands, as tonsils, parotids, submaxillary and sublingual glands, but frequently of no great importance. A few, from an internal tumour, have had a large external œdematous swelling of the subcutaneous and cellular tunic, from the chin down to the thyroid gland, and up the side of the face. Not a few early in the disorder have had gangrenous sloughs formed in their mouths, and perhaps so early in some, that the disorder was scarce complained of, till the slough was formed, so quick has it been in its progress." And again: "I have not mentioned a fœtor oris, because though some have had it, others have had it not." The symptoms of its extension to the larynx are then given, and he goes on to say: "I have frequently examined the matter these patients have at times spit; the greatest part was of a jelly-like nature, glairy, and somewhat transparent, mixed with a white opaque thready matter, sometimes more, sometimes less, resembling a rotten membranous body, or slough. Such a slough I have seen generated on the skin of one of these patients in the neck and arm, where blisters had been before applied. . . . . This white surface had the aspect of an oversoaked membrane, which, being oversoaked, had become absolutely rotten. The part blistered, if not quite, was in effect dry, and the flux from the slough was incredibly great. . . . . I scratched the slough with my nail; it separated with ease, and without being felt by the child. What my nail took off afforded the same appearance with the matter of the spittle before mentioned. Hence I thought I saw sufficient reason to convince me that the disorder in the larynx and aspera arteria was similar to this, generated in the same manner, and arising from the same internal cause; . . . . and it is likely, had the anatomical knife been employed, that what was seen on the back of one, might have been discovered in the arteria aspera of the other. There is a circumstance which adds to the probability of this opinion, viz. in one or more instances these different disorders appeared in different subjects in the same family at the same time."

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[1] Phil. Transactions, vol. xlvi. p. 435.

[2] An Account of the Sore-Throat attended with Ulcers, by John Fothergill, M.D. 8vo. Lond. 1748. 2d Edition.

[3] Phil. Transactions, vol. xlix. pt. 1.

[4] De Morbo Strangulatorio Opus, Ætii Cleti, Signini. 8vo. Romæ, 1636.

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[1] Loc. cit.

A century again elapsed before the disease attained to epidemic intensity: during the interval, outbreaks of more or less extent and severity occurred in various parts of France, Germany, North America, and in England, Scotland, and Ireland; sometimes sufficiently serious to receive full comment in the medical periodicals, sometimes so isolated and rare as almost to escape notice.

Paris, at the beginning of the present century, was a frequent seat of its appearance, and the death from this cause of a promising member of the Imperial family of France in that city, in the year 1807, was the occasion of a large share of medical attention being directed to its elucidation. No real progress however was made towards that object until the epidemic at Tours, in 1818, received a thorough investigation under Bretonneau. From this time the *Archives Générales de Médecine* contain frequent reports of its appearance in various parts of France, and, by recording its period of greater or less prevalence in Paris, and the details observed in the Hôpital des Enfants Malades, afford valuable contributions to the study of the disease: most of these notices are to be found under the heads of Croup and Epidemic Croup.

The records of the disease in this country become less frequent after the close of the last century.

Dr. Mackenzie, of Glasgow, describes two cases that came under his notice in 1813, of throat-disease ending in croup, and says that this kind of disease was very prevalent in Glasgow in 1819; he gives a careful description of it, agreeing very closely with the results arrived at by Bretonneau: these, made known in England by the publication of his work in 1826,[1] rendered the differentiation of the two diseases possible, and henceforth they are described apart. Dr. Abercrombie[2] alludes to cases of this kind in Dublin; he says, though not a common affection in Scotland, yet that it was very frequent and fatal among children in Edinburgh in the year 1826, and that "it is evidently an affection quite distinct from the idiopathic inflammation of the membrane of the larynx, to which we commonly apply the name of croup." Dr. Webster, of Dulwich,[3] records the deaths of two children in one family, in a house near London, in 1824, and of four other cases of ulcerated sore-throat, involving the larynx, coming under his observation shortly after; they "all had," he says, "an assemblage of similar symptoms, and the common point of danger was the windpipe."

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[1] Des Inflammations Spéciales du Tissu Muqueux, et en particulier de la Diphthérite, ou Inflammation pelliculaire, connue sous le nom de Croup, d'Angine Maligne, d'Angine Gangréneuse, &c. 8vo. Paris, 1826.

[2] Abercrombie, John, M.D. Pathological and Practical Researches on Diseases of the Stomach, Intestinal Canal, Liver, and other Viscera of the Abdomen. 8vo. Edin. 1828. Pp. 53–56.

[3] The Institute, vol. i. p. 100. These cases were published in an Appendix to Mr. Higginbottom's Essay on the use of Nitrate of Silver. 8vo. Lond. 1829. Pp. 185—196.

Mr. Ryland[1] describes cases of the kind as epidemic in Birmingham, in the year 1837. Dr. Humphry, of Cambridge, observed a case in the Norwich Hospital in the same year, and Sir William Jenner[2] remarks, in his Lectures on Diphtheria, "I have seen cases of it every now and then as long as I have practised medicine."

Though isolated cases were from time to time observed, any memory of its epidemic violence in this country had almost passed away when its reappearance in South Wales was announced by Mr. J. D. Brown,[3] of Haverfordwest; about two hundred cases occurred there in 1849–50, of which forty were fatal. Some general conditions, which at present we are unable sufficiently to appreciate, including the effects of the two cold winters of 1853 and 1854, and the intense cold of January 1855, seem at this time to have checked its epidemic progress, and also to have stayed the ravages of cholera in this country. Traces of it meanwhile are to be found in other countries: Denmark,[4] Germany, France, the North of Africa, Madeira, Teneriffe, America, and Hindostan. Dr. Jackson[5] reports two cases occurring in Calcutta in 1853, one at a later period, and also thirteen cases at the Martinière school, five of which died. We have accounts of it at Lyons in 1851, at Avignon[6] in 1853, of its great increase in Paris[7] in 1852–53, and of its reigning simultaneously there and in many parts of France from 1855 to 1857, as a most fatal epidemic. In Boulogne alone it was the cause of 366[8] deaths in that period. It was present, though not extensively, in the armies in the Crimea[9] in 1855, and also at Moscow[10] in the same year, and in Algeria[11] the year following. It was epidemic in California[12] in 1856–7, and somewhat later in the Northern States of America.[13]

The epidemic attained its height in England in the years 1858 and 1859, and during these two years we may estimate that 20,000 deaths were occasioned by it. In 1860 the number of deaths had fallen from near 10,000 to 5,202, and though they have not since reached the latter figure, yet the mortality from this cause in London was almost as great, and in the north-western counties quite as great, in 1862 as in 1859.

In Scotland, where the disease prevailed at the same time as in England, the mortality from this cause did not reach its highest until

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[1] Treatise on the Diseases and Injuries of the Larynx and Trachea, by Fredk. Ryland. 8vo. Lond. 1837.

[2] Jenner, Sir W., M.D. F.R.S. Diphtheria, its Symptoms and Treatment. Lond. 1861. P. 3.

[3] Med. Times and Gazette, 1850, vol. i. p. 670.

[4] Beck in Oppenheim's Zeitschrift, b. xliv. s. 200.

[5] British Medical Journal, 1859, p. 373.

[6] Archives Générales de Médecine, s. 5, t. vii. p. 338.

[7] Ibid. s. 5, t. v. p. 260.

[8] On Diphtheria, by E. H. Greenhow, M.D. Lond. 1860. P. 68.

[9] Haspel, Gazette Médicale, 1855, p. 829.

[10] Tarassenkoff, Diphtheritis Epidemica, Med. Zeit. Russlands, p. 92.

[11] Notes sur la Diphthérite. Recueil de Mémoires de Médecine Militaires, s. 2, t. xvii. p. 392.

[12] Fourgeaud, Diphtheria. 8vo. Sacramento, 1858.

[13] Boston Medical Journal, vol. lix. p. 252.

1861. It is somewhat remarkable that though Diphtheria existed both in India and California, we have no history of any outbreak of it in Australia until 1859, when Mr. James Moore[1] records nine deaths from this cause, and the occurrence of 275 cases at the same time in New Norfolk, Tasmania. This part of the world is perhaps more exclusively in communication with England than any other. The appearance of the disease there is not until after it had attained in this country to its full epidemic development: moreover, though epidemic in North America, both in California and the United States, as early as 1857, it was not till 1859 that it made its appearance in Nova Scotia, the part of that continent in closest communication with this country.

The severity of the outbreak at Boulogne, the constant communication from our own shores to that place, and the number of English visitors and residents there, many of whom fell victims to the epidemic, excited reasonable alarm, not only of the advance of the disease, but of its direct introduction into this country through the medium of those affected. Individual cases were imported, both at Folkestone[2] and Dover, without the disease spreading in those localities; its independent appearance in Wales, Lincolnshire, Cornwall, and Staffordshire precludes Boulogne from being considered its origin; yet sources of infection imported thence may have afforded new centres for the propagation of the disease, and have contributed in some degree to the determination of its type.

ETIOLOGY.—The records of the first half of the epidemic in England are imperfect; many deaths from this cause were included in the registration under the heads of scarlet fever, croup, and cynanche maligna. A table,[3] published by Dr. Farr, in his letter to the Registrar-General, shows that, while in 1855–57 there are 1,846 deaths registered as cynanche maligna, and only 725 as Diphtheria, in the year 1858, upon the first introduction of the new general term,[4] the numbers are 1,770 as cynanche maligna, and 4,836 as Diphtheria; and that in the year following, when not only the name, but also the nature of the disease was more generally understood, 9,587 deaths are registered as Diphtheria, and only 597 as cynanche maligna. In looking through the returns under croup and scarlet fever, the two diseases most likely to be mistaken for Diphtheria, it is not difficult to infer, both from the unprecedented numbers returned under these heads, as well as from the usual number of deaths of the two sexes from these causes being reversed, that some other disease has been included, and this variation occurs in the very districts in which we have other evidence of Diphtheria being present: thus in Wales, while the deaths from croup in 1853 were 207 males and

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[1] Australian Medical Journal, July 1859.

[2] E. H. Greenhow, M.D. op. cit. p. 69.

[3] Twenty-fifth Annual Report, p. 178.

[4] This was in the first Quarterly Report of the Registrar-General, No. 37.

197 females, total 404; for the year 1854 they were 215 males and 298 females, total 513; they then continue at or near 500, till Diphtheria is introduced into the registration nomenclature, when they suddenly fall to 424. The same exceptional proportion is first noticed in the Cornwall district in 1854; in the three following years the whole number is doubled, and in 1859 undergoes the same sudden diminution. The west-midland, north-midland, and eastern districts, from which at this time we have the most frequent accounts of Diphtheria, show the same gradual increase, and the same sudden fall; and it cannot be doubted that of the 6,220 deaths returned as croup in the year 1858, at least 1,000 were Diphtheria. In scarlet fever this is still more conspicuous; the number of deaths mounts up from 14,229 in 1857 to 30,317 in 1858; and in the latter year, for the only time that I am aware of, the number of deaths of females from that cause exceeds the number of deaths of males. An epidemic of scarlet fever certainly accompanied that of Diphtheria, and culminated in the following year, when the deaths from it, Diphtheria being excluded, reached 19,907; so that we may reasonably suppose that of the 30,317 deaths registered under this head in 1858, near upon 10,000 were really owing to Diphtheria. The probability is that some deaths from this cause in previous years were so registered; on turning to South Wales in 1857, we find that of 267 deaths from croup, 177 were of females; and of 349 from scarlet fever, 185 were of females; so that in 600 deaths, chiefly of children, the majority of which ought to be among males, we find an excess of 40 among females.

In the second half of the epidemic, taking the four years 1859–62, the number of deaths registered from Diphtheria is 24,219; of these 11,229 are of males, 12,990 of females. Half these occur in the first five years of childhood, and show a nearly uniform fatality in each of these years, the first year only being somewhat below 10 per cent. of the whole number; half the remaining number, or 25 per cent. of the whole, take place between the ages of five and ten years, and about 10 per cent. during the next five years; in the next ten years, that is, from the ages of fifteen to twenty-five, the proportion of deaths does not exceed 6 per cent. of the whole; from twenty-five to thirty-five it hardly reaches 2 per cent. and is below 1 per cent. in a decreasing ratio for each succeeding decade. The proportional mortality from Diphtheria for each age, to the deaths from all causes, is :—

	1st Year.	2d Year.	3d Year.	4th Year.	5th Year.	5—10 Years.	10—15 Years.	15—25 Years.	25—35 Years.
Both Sexes	·5	2·	3·7	6·	8·	9·	6·3	2·5	·32
Females	·5	2·1	4·	6·4	8·8	10·4	7·3	1·8	·45
Males	·5	1·9	3·4	5·6	7·3	7·7	5·3	1·2	·2

These results are opposed to some statements with respect to the liability of sex, but fully bear out Fothergill's remarks on this subject.

He says:[1] "Children and young people are more exposed to it than adults; a greater number of girls have it than boys; more women than men; and the infirm of either sex are more likely to have the disease and suffer from it than the healthy and vigorous. If it break out in a family, all the children are commonly affected by it, if the healthy are not kept apart from the sick; and such adults as are frequently with them, and receive their breath near at hand, often undergo the same disease." He also remarks that "this disease has now been with us several years, and has consequently survived the different seasons and all the variety of weather to which we are subject."

Climate and season do not influence the nature of the disease; it presents the same features in Sweden as in Spain; nor is any difference observable in its appearance in our own humid climate from that which it presented to Aretæus, when he attributed its causation to the peculiarly dry air of Egypt. Hot and dry climates or seasons, the germ being present, seem to favour its development. Yet difference of season has no marked effect on the course of the disease, nor on its prevalence,[2] over limited areas; nor has a sudden change of weather any effect in originating or arresting its epidemic appearance.[3]

The London Quarterly Reports illustrate, not the influence of season upon the disease, but the variableness of its prevalence in the metropolis. On the commencement of the registration of Diphtheria in the second quarter of 1859, the number of deaths from this cause was 173, in the third quarter 190, and in the winter quarter 141. In the next year the third quarter shows the lowest, and the winter quarter the highest mortality. For 1861 the numbers are: 1st quarter, 139; 2d, 159; 3d, 168; 4th, 231[4]—a progressive increase rarely met with even in other epidemic diseases. The extreme degree of cold reached in the winter of 1860 had no influence in checking the second advance of the disease in London: that it was not the occasion of the increase is shown by the comparatively low number for the first quarter of 1861, and the very high return for the last quarter of that year, when the season was unusually mild.

The influence of contagion is established; not the exact mode in which it operates. The matter of infection would seem not to be capable of any wide diffusion in the air, but to cling to particular places, houses, and even chambers; hence it not only becomes endemic, but might also be distinguished as hypodemic, as well as epidemic. When epidemic, no kind of soil or situation influences its occurrence; of places in close proximity some escape, while others suffer, and then frequently some direct communication is to be traced with the districts previously affected: isolated cases are, however, frequently met with without any widely-spread consequences. The earlier part of any local epidemic is generally attended with the largest proportion of fatal results: during the period of greatest epidemic prevalence the disease is found to be subsiding in one district while advancing in another; the height of the epidemic coincides with the greatest number of local outbreaks. No general atmospheric condition can then be its cause, and whatever the unknown assemblage of circumstances that favour the progress of the disease, they would not occasion it without the presence of its own special reproductive germ; so that the idea of the epidemic constitution must always include that of contagion.

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[1] Op. cit. p. 30.
[2] Boudet, Arch. Gén. de Médecine, 1842, s. 3, t. xiii. pp. 144, 446.
[3] Gazette Médicale de Paris, 1848.
[4] Some deaths from this cause are included under Croup. *Vide* p. 77.

The theory of direct contagion, as set forth by Bretonneau, requires considerable modification; the possibility even of inoculation is rendered doubtful by the experiments of Trousseau upon himself, and of Dr. Harley[1] upon animals; nor is there reason to suppose that greater power of propagation attaches itself to the fibrinous exudation, or special product of the disease, than to some of the secretions and exhalations from the sick. I know of no instance of the disease being carried from one house to another by the passage to and fro of those who were themselves unaffected; the presence of one sick person in a house is sufficient for its communication to the susceptible, however carefully kept apart, so that the infectious matter must, to some extent, be diffusible in the air, and the danger of infection has seemed to me to be greater from this source than from direct contagion. Most of our brethren can speak of having come into disagreeable contact with the secretion from the throat of sufferers, no bad effects following: very many can refer to dangerous illness in themselves as the result of too long or too close attendance in the sick-room. It is probably to this influence rather than to direct inoculation that is to be attributed the death of Valleix, and of Dr. Adams of Boston, U.S.[2] The case of M. Herpin, adduced by Bretonneau, admits of the same explanation: it is not clear that a very limited denudation of the skin, or of the mucous membrane, renders the system more exposed to the contagion; but the disease once contracted, those parts of the mucous membrane exposed to the air are specially liable to be affected by the disease; or if there be any abrasion of the skin, it is likely to suffer.[3] The existence of excoriation, as at the edge of the nostril, of unhealthy gums, of enlarged tonsils, possibly of catarrh, may determine the local manifestation of the disease without these lesions having been the channel of its admission.

It has been noticed that the more abundant the secretion in particular cases of Diphtheria, the greater has been the consequent extension of the disease, especially where the greatest care has not been given to cleanliness; even where this has received every attention infection will sometimes cling with surprising tenacity to particular houses or apartments. A single case, in itself not severe, occurring in a house, is sufficient to originate the severer form of the disease in the susceptible. It is not necessary that there should be much secretion from the mouth and nares to originate infection; a man named Sheppard, under Dr. Reynolds' care in University College Hospital, 1865, for paralysis subsequent to Diphtheria, contracted the disease from his child whom he held constantly in his arms during a short illness, the nature of which was not evident until after the child's death from asphyxia. A very short time suffices for the development of the disease after exposure to infection; the boy in the Hospital for Sick Children whose case is reported by Sir W. Jenner,[1] was observed to have advanced symptoms of Diphtheria within thirty hours of the commencement of the affection in a child occupying the next bed, and it is probable that a time considerably shorter is sufficient.

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[1] Transactions of Path. Soc. of London, vol. x. p. 315.

[2] Med. Times and Gazette, 1861, vol. i. p. 375.

[3] Mr. Lister, while House Surgeon at University College, received a scratch on the finger while performing tracheotomy in a case of diphtheria. Signs of the disease subsequently appeared in the injured part.

Evidence of infection is not always shown immediately after exposure; of two children removed from an infected house to a village where no case of Diphtheria had occurred, the elder showed the first symptoms of the disease on the third day, having been very cheerful and well the whole of the two intervening days. A young lady going from this village to nurse her sister in the infected house, became ill four days after her arrival. Eight days is the longest interval that has been known to occur before the disease has shown itself where prompt removal from the source of infection has been carried out.[2] Mr. W. Adams, of Harrington Square, has kindly furnished me with the clearest particulars of the case in point, mentioned at page 46 of Sir W. Jenner's book. A much longer interval may occur where the cause of infection is acting more continuously. Dr. Hughes,[3] of Denbigh, records a case where a servant was taken ill twenty-two days after the occurrence of Diphtheria in the house where she was residing. In a vicarage in the country a servant was ill with Diphtheria, and remained one month in the house till convalescence enabled her to be removed; an interval of ten days was then allowed for cleansing and ventilating the house, when the family, consisting of mother, a weaned infant, and two other children, the eldest under four years of age, who had left the house on the first appearance of the disease, returned home; three weeks afterwards the infant had symptoms of Diphtheria, and before the death of this child, which took place on the tenth day, the mother who had been constantly in attendance was taken ill, and subsequently the two other children.

Infection may be disseminated for an uncertain time by those convalescent from Diphtheria: a girl ten years of age was removed from home at the outbreak of Diphtheria, from which three children, two younger and one older than herself, afterwards died; two other sisters convalescent from the disease went to the sea-side, the one five weeks from the commencement of her illness, the other only three weeks, but from whom the last trace of deposit had cleared away; they left together and seemed to be equally well: they were joined by other members of the family, and at the end of the week by the little girl, æt. 10: early in the following week symptoms of the disease appeared in her, and became rapidly fatal. She was the only one of the family that had been isolated during the whole period of the illness. Sir W. Jenner[1] gives two similar instances: in the first, only a fortnight elapsed from the commencement of the disease to removal into the country, and within a fortnight from the date of arrival, another member of the family, some time resident there, was attacked; in the second a little boy, after three weeks' separation from his family, joined his sisters, convalescent from Diphtheria, at a country residence to which on their recovery they had been removed. "Ten days after his arrival there, the boy sickened with Diphtheria. In this case, either the poison was in the child's system when he left London, and remained latent for a month, a supposition highly improbable, or he caught the disease from his sister, after they met in the country."[2]

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[1] Loc. cit. p. 83.

[2] Exactly this interval has been observed under the same circumstances in scarlet fever, as the longest that has come under my notice.

[3] British Medical Journal, 1859, p. 80.

Constitutional predisposition has a great influence in increasing the liability of families and individuals to receive the disease: of two families residing in the same house several members of the one have suffered, while all of the other have escaped; a difference of susceptibility is also observed in members of the same family, and this not always in favour of the seemingly more robust. Allowing for the effects of similarity of diet, occupation, general management and hygiene, and chiefly for the degree of exposure to the same sources of infection, it is yet impossible to contest the effect of family constitution, both in favouring the occurrence of the disease, and in disposing to its fatal termination.

Great mental activity, and a high degree of nervous susceptibility, would seem to increase the liability to become affected; bodily fatigue, and exhaustion from any causes, predispose. There appears to be but little difference in the liability of the different classes of society.

The presence of other diseases, as of typhoid fever, pneumonia, measles, erysipelas, and scarlet fever, has a great effect in exposing the system to the attacks of this one. The special poison of this disease sometimes produces effects short of general Diphtheria, which, however, predispose to the occurrence of the fully-developed disease, either from the first local evidence of it being unchecked, or upon some accidental source of weakness arising, as from the effects of aperient medicine, or of menstruation. The disease is especially likely to be established, if there be repeated or continual exposure to the exciting cause.

The recurrence of Diphtheria more than once in the same subject, is not settled so conclusively in the affirmative as has been supposed. That the same person may repeatedly suffer from the slighter forms of the malady, and that some do so upon the slightest exposure, is frequently observed; but when the fully-formed disease has been undergone, though relapses are to be feared in convalescence, even during the whole of the subsequent period of debility, which may be prolonged for two or three months, independent recurrence is rare. Of the children that recovered from the disease at the commencement of the epidemic, and who have been constantly under my observation since, no instance of a second visitation has occurred. Such instances are recorded, one by Dr. Gull, eleven months after the first attack, and three others by Dr. Greenhow,[1] one of which was fatal, but no particulars are given in this case as to the intensity of the first attack. The development of Diphtheria agrees more closely with that of erysipelas than of scarlet fever; the closeness with which many of its pathological effects coincide with those of the latter disease, makes it necessary to remark here, that the occurrence of scarlet fever offers no protection against the attacks of Diphtheria.[2] So with respect to croup: two instances have occurred to me, wherein children that had suffered severe attacks of croup, fell victims to Diphtheria after intervals of four and six years. Hygienic conditions influence the frequency, and even the fatality of Diphtheria, less directly than they do the permanence and diffusion of the infectious principle; want of cleanliness allows the infectious particles to accumulate; the presence of decomposing organic matters shields them from destructive oxidation in the atmosphere; thus they linger in districts naturally malarious, and in those artificially so from an improper drain system, as well as in the close, dark, and dirty districts of large towns, and evils thus matured rest not in their cradles, but come forth and often exert their most fatal effects in homes the most unlike those where negligence had allowed them to lurk.

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[1] Loc. cit. p. 52.

[2] Loc. cit. p. 53.

Symptoms.—Some general constitutional symptoms precede those occasioned by the concomitant local changes; both may co-exist in every degree of severity, sometimes the one, sometimes the other attaining a deceptive prominence; at other times both are so obscure as to make the inroad of the disease remarkably insidious. Among the earlier symptoms are yawning or sighing, shallow and infrequent respiration, great lassitude and debility, some aching of the back and legs, either a distinct rigor, or chilliness, pallor, a sense of nausea or rising in the throat, anorexia, sometimes vomiting, or diarrhœa, and in children convulsions, a sense of constriction across the forehead, or intense headache, vertigo, extreme muscular weakness, some anomalous fixed pain, an altered mental state, slowness of recollection, an indifference of manner, and an obtuseness of the mental faculties; this latter gives place to a short period of excitement, during which it may be thought that no illness is impending. There is, however, a marked elevation of temperature, which is increased at night, when wakefulness or restlessness always occurs. The pulse is accelerated, and in children or young persons may rise to 130 or even to 140 in the minute: this frequency soon subsides, generally before the end of the second day; and though the pulse continues to be quick, it is either feeble or easily compressible. The respiration is never proportionately accelerated at this period. The tongue is moist, with a thin creamy fur; the urine is pale in colour, at first free, soon rather less in quantity, with scanty deposit of lithates, but still pale, and at this time it may be albuminous. The lymphatic glands at the angle of the jaw are already perceptible; there is always some enlargement of the cervical glands, and redness with a little swelling of the posterior part of the soft palate, of the fauces, of the back of the pharynx, and of the tonsils. The throat is sore, deglutition is difficult if not painful, sometimes the pain is felt in the ear, and there is frequently stiffness of the neck; very little external swelling is at this time noticeable, and there is no diffused redness of the skin; sometimes little isolated red spots are found in different parts of the surface, as over the neck or behind the ears; there may be suffusion of the eyes and slight injection of the conjunctival vessels, and a little obstruction to the nasal passage from a similar vascular state of its lining membrane. Sometimes this is seen at the nares to be simply red, sometimes an opaque white spot may be noticed beneath the epithelium, or one nostril may be completely obstructed, and some fibrinous exudation already observable; sometimes the back of the pharynx will show the first indication of this in lines of opaque tenacious secretion, or commencing deposit is seen there, in the apices of its enlarged follicles. The earliest evidence of the disease is, however, most frequently found within the follicles of the tonsils or deposited on its inflamed and turgid surface.

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[1] Loc. cit. p. 111.

[2] Dr. Ballard, in Med. Times and Gazette, 1859, vol. ii. p. 78.

The first general symptoms are transient and may be so trifling in degree as to escape notice, until they are intensified by the progress of the local lesion; this will give rise to pain, heat, and soreness of the throat, with impeded function: it also excites some sympathetic febrile disturbances of its own, and always increases that proper to the general disease; where both are severe, the throat, though covered with exudation, is often the least part of the patient's complaint; where both are slight, there may be an interval in which little complaint is made. During this interval, two days, there may be no visible exudation in the fauces; the tonsils continue to be enlarged and their surfaces irregular, and they in common with the whole of the pharynx, the arches of the palate, the velum, and the uvula, are of a deep-red colour and unequally turgid; one side is generally the most affected, the uvula is enlarged, red, and glistening, and a mottled redness extends forwards from it over the soft palate, but the rest of the buccal membrane is pale. Some of these parts soon appear more tumid and glistening than others, and spots at first semi-transparent, and afterwards opaque, rapidly form and coalesce, so that in a few hours a large surface may be covered with a continuous layer of exudation; or the exudation is limited to one or more centres, the mucous membrane around being elevated and of a violet tinge of redness, until it is invaded by the same change. The raised edge is then extended, the centre being occupied by a flattened, yellowish-white, leather-like deposit, increasing in thickness by additions to its under-surface; this is accompanied by the pouring out of a considerable amount of a more fluid secretion, while that from the surrounding mucous follicles is also increased and altered in quality, so that a tenacious fibrinous matter is mingled with the mucus. The cervical glands increase in volume, the submaxillary lymphatic glands especially; a large amount of serous infiltration takes place in the surrounding cellular tissue, and the whole of the front of the neck becomes greatly swollen: deglutition is involuntarily suspended, secretions escape in some quantity from the mouth, and even from the nostrils, by which the lips are excoriated. The superficial layers of the localised deposit, partially separated and exposed to the passage and re-passage of the air, and the warmth and moisture of the mouth, undergo decomposition and give rise to great fœtor; extravasated blood mingles with the decomposing exudation, or escapes freely from its under-surface, or occasions epistaxis. The voice is muffled or nasal in tone; impeded respiration may occur from the physical obstacles in the fauces without laryngeal symptoms arising; or these being slightly marked, symptoms of pulmonary obstruction come on insidiously. These processes may be gone through with great rapidity, and are accompanied by the most marked adynamia; the pain in the head is intense, the restlessness and agitation extreme, vomiting or diarrhœa may occur, the lips and tongue become dry and brown, the pulse very rapid, feeble, and irregular; the skin becomes cold, the face pallid, the whole attitude is indicative of powerless exhaustion; there is impaired consciousness, slight delirium, or deep somnolency, and some repeated attack of syncope terminates life.

The elevation of temperature is always marked, and characteristic: it may be as high as 103° very early in the disease; it will generally reach this point by the end of the third day, and is highest at night, when it may amount to 104°, then probably with delirium. This temperature has not been exceeded in uncomplicated cases. The days of highest temperature observed from the commencement of the illness have been from the third to the nine or tenth. Free exudation is attended with temporary depression of temperature. Defervescence in the severer cases recovered from has been on the twelfth and fourteenth days; in the milder cases this has not occurred before the sixth day, though some precedent subsidence of temperature may have been noticed. In fatal cases an earlier decline of temperature has sometimes been met with. A girl, aged 8, had a surface temperature of 103° on the third day, 101° on the fourth, then barely 100°, with swelling of glands, fœtorosis, and hæmorrhagic tendency, death on the eighth day; the day before the temperature in the axilla was below the normal. In cases markedly asthenic the temperature of the surface falls, while that of the interior of the body continues to be high: in a boy, aged 2, with a temperature of 103° in the rectum on the fifth, sixth, and seventh days of the disease, only 100° could be obtained in the axilla, and sometimes only $99\frac{1}{2}°$; the temperature after subsiding to 100° was suddenly raised to 102° by acute kidney complication; continued there two days, and was again below 100° the day before death. Another boy, aged 4, with nasal diphtheria and slight tracheal implication, had a temperature of 103° in the rectum on the sixth day, suddenly falling to 99°·5 on the seventh, then continuing at or near 100° until death on the fifteenth day. In the apparent interval between the first illness and the distress occasioned by the throat symptoms, the high temperature is an indication of value: a child has been taken out for a walk on the fifth day with a temperature *in recto* of 103°·7. A young man was found dressed and walking about on the ninth day of illness, with a surface temperature of 102°; on being kept in bed the temperature fell two degrees in two days; the exudation then ceased to extend, but the tonsil last affected had become turgid and more inflamed coincidently with the fall of temperature: albuminuria appearing here during defervescence was considered to be rather hæmic than renal.

During convalescence the temperature is low, and readily depressed: but any intercurrent affection may keep up the temperature indefinitely.

The advancing asthenia is always accompanied by a considerable increase of exudation; it continues while the process of exudation is going on, and is at its greatest when the separation of the diseased products is being accomplished: this may be completed by the ninth or tenth day of the disease. The exhaustion at this period, if not fatal, is often so great as to suspend for some days the hopes of recovery, to delay the process of repair, and to prolong the diseased action, so that there may be a reappearance of exudation in the fauces, or it may at this time invade the larynx. If there be any injury of the skin, the diphtheritic action, probably already established there, will be continued so as to become an additional source of exhaustion. Besides the hæmorrhagic tendency at any affected surface, spots of purpura sometimes make their appearance either widely distributed or grouped together; sometimes an earthy pallor pervades the whole surface of the skin. Where there are none of these unfavourable complications it is not rare to find that, when the exudation is clearing off from the fauces, a remarkable increase of albumen occurs in the urine. Nocturnal delirium may occur, not only at the commencement, but at the height of the disease, when the asthenia is considerable. A more violent symptomatic disturbance at the commencement of the disease, occasioned either by local or by constitutional reaction, may mask the tendency to asthenia without making it less; and should the course of the disease not be interrupted by the implication of the air tubes, asthenia becomes the prevailing condition throughout all the subsequent illness, and may bring on a fatal result, not only at the end of the second week, but during any of the subsequent weeks of convalescence, even to the fifth or sixth from the commencement of the disease.

Extension of the disease to the larynx and trachea may be the cause of death at a much earlier period: the air-passages are specially liable to become the seat of the peculiar exudation of Diphtheria; it may commence there either by independent centres of deposit, or by the spread of exudation from the pharynx to the epiglottis, over the arytæno-epiglottidean fold, and thence downwards, even to the remotest bronchi, and be fatal, with symptoms of sudden or of slow suffocation, in the earliest stages of the disease, or at the period of greatest exudation, towards the end of the first or commencement of the second week. This extension often proceeds insidiously when the general symptoms are of great intensity, and is only indicated by hoarseness, or weakness of the voice, and by some laryngeal quality in the breath sounds, faintly audible during both inspiration and expiration, by signs of impeded respiration, evidenced in the œdema of the face and livid hue of the lips, sometimes only by pallor, and gradually deepening unconsciousness, and by the retraction of the softer parts of the thoracic parietes. In other cases the first invasion of the mucous membrane of the upper part of the air tube is announced by highly characteristic phenomena, similar to those produced by acute laryngitis in adults, or by idiopathic croup in children, differing only in the less sharp and sonorous clang of the cough, in the more husky tone of the voice, and in the antecedent and concomitant symptoms. Besides the appearance in the fauces, and the dysphagia, the sudden change from the asthenic character of the prevalent symptoms to the excitement, arterial and general, now occasioned, is very striking. In other cases, chiefly among children, the disease may have set in with no great severity, the power of swallowing and even the desire for food may be returning, and the child, no longer confined to bed, is resuming its amusements, when a hoarseness of voice and a noise in breathing are the only precursors of paroxysmal dyspnœa, differing only from that of croup in the time of the day in which the first attack may occur, and in the readiness with which the child will return to play on its subsidence, but soon becoming more terrible in the unintermitting violence which it rapidly assumes, or in the suddenness of the fatal result.

The mortality from this complication alone is very great; it has been estimated that one-half of the fatal cases of Diphtheria die from this accident; nor is this estimate excessive when children are the sufferers; an extension to the air tubes, necessarily fatal to children, will sometimes be survived by adults: in the man Sheppard a cough of unequalled violence with expectoration of false membrane continued into the third week of illness. In a small proportion of cases the larynx or trachea has been the first seat of the disease; in these cases and in those where the faucial deposit is small or already disappearing, the urine is more frequently found to contain albumen at an early period than in those cases attended with abundant exudation on the pharynx. In a female, aged 35, named Bowra, under Dr. Hare's care in University College Hospital for Diphtheria, in April 1865, who died of pneumonia, coincident with slight deposit in the larynx and fauces, there was a considerable amount of albumen in the urine on her admission, the second day of her illness; the next day the temperature of the surface rose to 104°, pulse 144, respiration 36; the albumen increased to one-sixth, and the respiration to 57; death took place on the sixth day. The first evidence of deposit was in the larynx, and there was no extension of it into the bronchi. Soreness in the front of the neck, and pain or sense of tightness over the sternum, are frequently complained of in these cases, and sometimes slight œdema is noticeable in front of the trachea.

Albumen is found in the urine in the great majority of cases; its presence either in large or small quantity does not necessarily affect the excretion of urea: this is always increased during the whole period of illness, and when the disease is at its height is frequently double. In one case (J. B., a young man, aged 18), on the seventh day of illness, 606 grains of urea were excreted, or more than treble the normal quantity: the urine has an acid reaction, a high specific gravity, and deposits a furfuraceous sediment in which uric acid, urates, oxalates, and sometimes phosphates, are detected by the microscope, and not infrequently casts of the renal tubules, either waxy or granular; blood-corpuscles are rarely found, and never in large quantity. Albuminuria generally occurs early in the illness; it may be absent and reappear more than once in its course. In a man named Walker, aged 42, admitted to University College Hospital, under Sir. W. Jenner's care, Nov. 14th, 1864, for Diphtheria, at the end of the first week of illness, there was no albumen found till the end of the third week; it then continued till death, which took place in the fifth week of the disease. In the case of J. B. albumen was present throughout the illness; on the twelfth day it had increased to one-third, on the twenty-fourth day it was one-eighth, and then gradually lessened during the ingress of paralytic symptoms, and finally disappeared at the end of the fifth week, while the nervous disorder was at its greatest, and three weeks before convalescence was established. Albuminuria is not persistent after recovery, and does not often result in anasarca.

Hæmorrhage is not infrequent, and may be so profuse as to cause sudden exhaustion, either on the separation of some morbid deposit of unusual depth, or from some dyscrasia of the blood favouring its ready transudation. Vomiting and diarrhœa, generally absent after the first ingress of the disease, may set in with gastralgia and great depression. Exhaustion may come on gradually without these symptoms, the pulse increases in rapidity and feebleness, and death takes place without distress of breathing or impairment of the mental faculties. In some of these cases, and of those yet to be described, fibrinous coagula form before death in the cavities of the heart.

The liability of other mucous membranes and of the skin, especially when denuded, to become the seat of the changes characteristic of Diphtheria, has often a great influence on the progress of the disease; eczema behind the ears, or in any fold of integument, abrasions or fissures at the juncture of skin and mucous membrane, leech-bites, blistered surfaces, and even those irritated by a mere rubefacient application, may become affected, and add greatly to the amount of disease against which the patient has to contend: a cutaneous eruption, occasioned by the disease itself, is sometimes, though rarely, the seat of these ulterior changes; the cuticle is raised, a white surface is exposed—this is identified with the upper layer of the cutis and does not implicate its whole depth—the skin immediately around is red and tumid; new vesicles, sometimes sanious, are raised upon it, they burst and coalesce; the white layer extends its surface, which is still somewhat below the level of the surrounding skin; it increases in thickness by additions from below, and the upper layers soften, decay, and disintegrate; it is accompanied by an irritant fluid secretion, both increase proportionally, and the extension of false membrane takes place most readily in the direction where the secretion is most in contact with the skin; where these changes take place in some portions of the skin, towards the end of the disease, there is but little secretion formed, and no great extension occurs. At the height of the disease the extension is sometimes very rapid; but this is less remarkable, because less constant, than the tendency then observed to repetition. Not only will every abraded or irritated surface take on this action, but isolated patches of deposit may occur on the edge of the lip, on the eyelid, in the meatus auditorius, and elsewhere. Open wounds undergo a similar change, the superficial granulations are converted into this pseudo-membranous layer, cicatrization stops, the wound becomes painful, and a copious ichorous secretion is poured out; a fissured nipple has been the starting-point of the morbid process, and much of the surface of the breast has suffered; the disease readily establishes itself in the vagina, where layer upon layer of false membrane may accumulate. An abraded surface of integument is sometimes covered with a granular or pulpy exudation only, attended with but little fluid secretion; the affected surfaces then continue to be irritable, but do not always extend. Diphtheria has generally first appeared in the fauces, and made some progress before other parts of the mucous membrane, or of the exposed skin, have been attacked; instances are not, however, wanting of the disease commencing elsewhere, and inducing in the pharynx, larynx, and trachea, the characteristic changes that soon become fatal.

A remarkable train of symptoms often make their appearance after the disease has reached its height, and become a new source of danger. These are the limited and varying series of paralyses, which are some of the characteristic effects of the special diphtheritic poison. An altered tone of voice and the regurgitation of fluids through the nose are frequent evidences of this, and have long attracted notice; an inability to swallow, first pointed out by Ghizi, was noticed by M. Guersant to occur about the ninth day of the disease, and to become an embarrassing obstacle to recovery in cases where tracheotomy had been performed. This is frequently observed only with respect to fluids; there is at this time, as shown by M. Trousseau, a loss of sensibility in the velum pendulum palati, and probably in the pharynx and glottis. Complete inability to swallow from loss of power in the muscles of deglutition seldom occurs before the third or fourth week of the illness; at this time, on inspection of the throat, the uvula is often found to be lax, and neither it nor the pillars of the fauces act upon stimulation; difficulty in the ingestion of sufficient nutriment adds to the dangers of this period; vomiting, moreover, is possible when the power of swallowing is gone. Loss of power and of sensibility in the parts supplied by the par vagum occur at a somewhat earlier period than the paralytic affections of other parts of the body, and hence arise special sources of danger; the slow weak pulse observed in the second and third weeks of illness is from this cause, and is frequently a fatal symptom. The pulse may be reduced in children to sixty or even forty beats in the minute, and the child seeming otherwise well, death by syncope has occurred suddenly, on some undue exertion; or the heart's pulsations have gradually failed to thirty or, shortly before death, even to sixteen beats in the minute.[1] I have seen a similar failure of respiration occur to a boy, aged 9, in the second week of illness, where no extension of the disease to the larynx had taken place; inspiratory efforts had to be artificially excited at frequent intervals during many hours; every few minutes the respiratory movements became more and more shallow and inefficient, until they were again stimulated into action, and even then the ribs were not always elevated; at the end of twenty hours respiration was more satisfactorily performed; during the second day the voice regained its clearness and force, the intellectual activity seemed to be more than usually quickened, the respiratory murmur was perfect in every part of the lung, both food and stimulant were swallowed, but the action of the heart became at first slow and weak, afterwards very feeble and rapid, and death took place on the third day from the commencement of these symptoms. Paralysis of the muscles of respiration occurring at a later period, in an adult, threatened to be fatal but for the stimulus of galvanism. Dr. Gull reports the case of a boy, aged 11, who, five weeks from the commencement of Diphtheria, was unable to prevent the head falling forward, or to either side, owing to paralysis of the muscles of the neck; he suffered dysphagia, aphonia, and paroxysmal dyspnœa; a few days afterwards "the breathing became entirely thoracic. The diaphragm was unmoved in inspiration and depressed in expiration, indicating a loss of power in the phrenic nerves."[2] Death approached rapidly by apnœa.

Loss of power and irregular action of the pharyngeal muscles is not only the earliest, but the most frequent form of disordered innervation. It sometimes rapidly disappears, and is not followed by other symptoms of this kind; in other cases the power of swallowing may continue to be impaired for three or four weeks; at first the difficulty is with liquids, afterwards it is now and then found that solids cannot be passed down, and are in danger of remaining in the lower part of the pharynx, or of being forced up towards the posterior nares, while liquids find their way into the stomach, and these conditions may alternate. At this time articulation is sometimes defective from imperfect movement of the tongue; tingling sensations in the tongue and lips are also felt, and may continue during the fifth and sixth weeks. Paralysis of the nerves of the special senses was first observed as a consequence of Diphtheria by J. F. Hoffman.[1] Taste is sometimes lost, more rarely hearing; defective vision is not infrequent; it begins with an immoveable and sluggish pupil, and an inability to read or to distinguish near objects; soon more distant objects become indistinct, double vision or strabismus may occur; sometimes one eye only is affected; these changes of vision take place from the fourth to the seventh week, and when sight is perfect the pupils again act freely. As these alterations of sense improve, numbness and tingling commence in the fingers and toes, extending gradually to the hands and arms, and to the feet and back of the legs, or even to the hips. Some degree of these sensations, with formication or a sense of coldness in the extremities, various degrees of anæsthesia, or even some hyperæsthesia, may occur earlier, and before any great loss of muscular power. These conditions vary very much, disappearing at some parts, increasing and extending at others. They may pass off for a time and reappear with the increasing muscular debility; the sense of touch may continue to be impaired; anæsthesia is seldom complete except in the severest cases. The lower extremities suffer most; it may be impossible not only to stand but to move the legs in bed. When this degree of paralysis is not reached, the gait is often unsteady, and walking may be impossible from a loss of the muscular sense; this sometimes interferes with the use of the hands, and the power of co-ordinating muscular movements. Loss of power is not to the same degree or so persistent in the upper extremity as in the lower, though here also remarkable alternations are observed, one limb or one set of muscles being free to act on one day, and powerless another; when power is regained, one muscle, or part of a muscle, may remain for a time the subject of inaction or of spasm. Reflex action is often diminished, but not increased. Paralysis of the bladder has occurred, not of the rectum. Constipation, from paralysis of the abdominal muscles, is a frequent condition demanding attention. The first symptoms of paralysis will almost always appear before the end of the fourth week; they generally attain their greatest degree of intensity by the seventh or eighth week; they may not do so until the tenth or twelfth, as in Sheppard's case; all traces of the affection may not have entirely disappeared after five or six months. Lesions of innervation are not in proportion to the extent and persistence of the local lesions, nor always the consequences of the more severe and prolonged attacks of Diphtheria only, though it is after these that they are most likely to occur; they may conduce not only indirectly, but directly, to a fatal result, from progressive loss of nervous power, apart from muscular weakness; in these cases the period most dangerous to life is reached in the seventh or eighth week. Trousseau[1] has detailed a case where delirium and convulsions at this time appeared, with ultimate recovery. More frequently death by asthenia, unattended by symptoms of other disease, occurs within this period. A peculiar pallor and opacity of the skin is often a concomitant of even the more slightly marked cases of paralysis consequent on Diphtheria. In the more prolonged cases there is always some evidence of anæmia.

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[1] Sir W. Jenner, op. cit. p. 44. Mr. Adams has given valuable details of a case of this kind in the Second Report of the Medical Officer of the Privy Council, p. 327.

[2] Lancet, 1858, vol. ii. p. 5.

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[1] Rust's Magazin, 1831, b. xxxiii. s. 241.

DIAGNOSIS.—A careful inspection of the fauces will, in the majority of cases, be conclusive as to the presence of the disease: where a yellowish patch of exudation moulded to the surface it has invaded is thus brought into view, the nature of the serious illness, which may have been obscure, is at once revealed: where this anatomical character of Diphtheria is not obvious, the appearances in the fauces, taken in conjunction with the associated phenomena, will still be of primary diagnostic value. It is important that the earliest stages of Diphtheria should be distinguished from catarrhal affections. The redness of the fauces in Diphtheria is more intense but less uniformly diffused than in catarrhal inflammation; the tonsils are more tumid, and one side is more affected than the other; the lymphatic glands at the angle of the jaw and beneath the sterno-mastoid are always enlarged: in children coryza may be present, the vascularity of the conjunctiva is then more like that observed in measles, but there is less secretion; afterwards, the defluxion from the nares becomes more considerable, it is not simply mucous, but sero-purulent or sanious; or the nares not being so much affected, a quantity of unequally opaque and tenacious mucus bubbles in the gullet, and prevents the view of the posterior part of the pharynx; there will be pain in deglutition, as shown by the infrequency of the effort, or the grimace that accompanies it; in either case the exudation soon extends to the larynx, and the cough, hitherto infrequent and moist, becomes frequent, dry, and croupy, and the disease is set down as catarrhal croup. For the further diagnosis of croup from Diphtheria, see the article on Croup.

Tonsilitis resembles Diphtheria in the two sides of the throat being unequally affected, and in the occurrence of external swelling at the angle of the jaw; the constitutional symptoms are, however, symptomatic, dependent on, and in proportion to, the local complaint; the lymphatic glands are not enlarged at the commencement of the attack, nor those beneath the sterno-mastoid at any time. A yellowish soft secretion appears at the orifices of the tonsil; the mucous surface preserves a smooth, glistening appearance, and any exudation of lymph upon it is semi-transparent, very thin, and limited in extent; the tendency of the inflammation is to resolution or suppuration, the other tonsil often becomes affected in the same way, but without any similar change taking place in the intermediate mucous membrane.

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[1] Gazette des Hôpitaux, 1860.

In Herpetic sore throat the highest temperature is on the second day, when it may rise to 102°, and then rapidly subside.

In scarlet fever, the throat affection is always preceded by symptoms of severe febrile disturbance, which are persistent, and in proportion not to the throat affection, but to the severity of the attack of fever which follows; the chilliness and headache may not be so marked, but the heat of skin is greater, and the pulse at once attains a high degree of frequency which it maintains during the first days of the illness, and until after the appearance of the rash, or the condition of the throat, has removed all doubt as to the nature of the disease. The premonitory symptoms in Diphtheria are sometimes not noticed in the severer cases, and when well marked do not always indicate the approach of the graver symptoms; should these follow, the frequency of the pulse during the first few days is not maintained, the respiration is shallow and not proportionally accelerated, and there is neither the continued high temperature nor the same pungent heat of skin. The diminished frequency and fulness of respiration at the outset of Diphtheria is often an indication of value when the disease is not yet fully developed. The cervical lymphatic glands are enlarged in both diseases. The redness of the throat in scarlet fever is uniformly diffused; on the second or third day it becomes very intense, appearing simultaneously upon all parts of the throat and palate, and affecting the papillæ of the tongue; both tonsils are equally enlarged. In Diphtheria the redness and turgescence are greatest in certain parts about to become the seat of exudation, and at the edges of the exudation already formed, so that it and the surrounding redness gradually advance upon the contiguous portions of the mucous membrane; the papillæ of the tongue are neither red nor enlarged, and the tonsils are unequally affected, In scarlet fever exudation, both tonsils are covered with a milk-white layer applied equally to the surfaces of both, and the soft palate and tongue may be covered with a similar layer; this undergoes no great increase in substance, and at a certain period is detached in shreds; it is not capable of absorption, and on its separation leaves a red and sensitive surface. In Diphtheria the process of exudation continues to be active for some time; on its cessation, there is a separation of membranous layers of considerable density and extent, which may represent a cast of the surface on which they formed; a re-absorption is now possible of some of the products still imbedded in the mucous tissue; this is accomplished either without loss of substance, or with superficial ulceration only, and the sensibility of the surface is diminished. In scarlet fever there may be sloughing of the tonsil, and there is a tendency to suppuration of the cervical glands. There is no tendency in the inflammation of the throat in scarlet fever to be propagated to the air-passages; the nasal tone of voice, and the regurgitation of fluid through the nose, cease on the subsidence of the swelling of the tonsils and lymphatic glands. In Diphtheria these symptoms often undergo a remarkable increase subsequently, from the paralytic sequels of the disease. Scarlet fever has a definite course, modified only in degree of severity; Diphtheria may either be arrested or modified in its course, so that the period of its duration is less definite. In scarlet fever there is a greater liability to inflammations of the serous membranes than in Diphtheria. Albuminuria, as a sequel to scarlet fever, interferes with the excreting power of the kidneys, is attended with hæmaturia, and more frequently results in dropsy and anasarca: it is an early symptom in Diphtheria, is rarely attended with hæmaturia, seldom interferes with the excretion of urea, and does not result in dropsy.

Both diseases are contagious, but while the contagion of Diphtheria has not been shown to give rise to scarlet fever, that of scarlet fever has apparently been followed by Diphtheria. They are not prophylactic of each other: in the late epidemic of Diphtheria, children who had gone through scarlet fever were equally liable to suffer;[1] and though in some cases a severe attack of Diphtheria has seemed to give an immunity from scarlet fever, yet instances are not wanting of those who have recovered from Diphtheria being attacked with scarlet fever. Dr. Buchanan,[2] of Glasgow, records the appearance of scarlet fever in a boy six years old, four days after the performance of tracheotomy, and in the second week of his illness from Diphtheria; anasarca appeared in the third week; in the sixth week convalescence was complete. A patient suffering from paralytic symptoms, consequent upon Diphtheria, while under the care of Dr. Stewart, in the Middlesex Hospital, contracted scarlet fever, during the febrile stage of which, and the full appearance of the rash, the paralytic symptoms cleared away. This modification of a symptom peculiar to the one disease upon the establishment of a condition peculiar to the other, though an instance of the distinct nature of the two, does not show any necessary antagonism between them; not only may the one succeed to the other at very short intervals, but it would seem to be possible for them to co-exist. The appearances in the throat characteristic of Diphtheria may commence after the subsidence of the redness occasioned by scarlet fever, and the disappearance of the rash; they may, however, come on at any period of its course, and more rarely the two diseases may seem to be coincident, so that the aspect of the throat on the first day may be indicative of Diphtheria, and with the characteristic rash of scarlatina appearing on the second or third day there may be a fall in the frequency of the pulse. In some epidemics of scarlet fever undoubted cases of the disease occur without the characteristic rash. A redness of the skin, and more or less marked rash or eruption, readily distinguishable from that of scarlet fever, has sometimes accompanied the early stages of Diphtheria. Mercatus[1] mentions a rash like flea-bites in some of the Spanish epidemics, and a redness of the whole face and neck, with loss of voice and dyspnœa. Fothergill describes a rash, in the first edition of his treatise, which he says, in a foot-note[2] in the second edition, did not regularly accompany the disease, and which is certainly not that of scarlet fever; it agrees more nearly with that lately observed, and described by Dr. Babington, as rubeola notha, which was sometimes, though rarely, seen in cases of Diphtheria, appearing on the first day. Dr. Fuller[3] communicated to the Harveian Medical Society, February 1858, the case of a child ill with sore throat and a rash, like scarlet fever, from the commencement; on the third day there were great dyspnœa and excitement with an increase of the rash and of the redness of the face; by night a membranous cast of the pharynx was expelled, with immediate relief to the dyspnœa and a rapid subsidence of the redness and rash, so that by the next day no trace of either remained; recovery was rapid, there were none of the sequelæ of scarlet fever, and no desquamation. Desquamation of the cuticle from the hands and feet has occurred after prolonged illness from Diphtheria, when there has been no preceding rash.

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[1] Dr. Ballard, loc. cit. p. 78.

[2] British Medical Journal, September 1864, p. 324.

Erysipelas comes on with rigors, or chills, and headache; there is pain in deglutition, often extreme, and some enlargement of the cervical glands; the throat redness, though intense, is diffused; there is no secretion, and none of the fibrinous exudation characteristic of Diphtheria: this form of sore throat may occur either before or after the appearance of the erysipelas on the face or head. Erysipelas, small-pox, and measles are liable to be followed or complicated by Diphtheria.

Diphtheria may follow upon typhoid fever or any prolonged and exhausting disease; it is important, therefore, to distinguish the sordes that collect in the fauces, and the special product of thrush, or muguet, to which such cases are liable, from the exudation of Diphtheria. The matter of thrush is closely attached to the mucous membrane when it is first exuded, but becomes more and more easily separable; it occurs in little rounded masses, is whitish, and soon projects beyond the level of the surface: if artificially removed, the membrane beneath looks slightly hollowed, and either red or grey, but it is neither completely abraded nor ulcerated; the buccal membrane and not the throat is specially the seat of this formation; it acquires no great extent, nor considerable tenacity. Chemically, it has an acid reaction, is not acted upon either by acetic acid or by alkalies, and is only dissolved or destroyed by sulphuric acid; these qualities are owing to the large parasitic vegetable growth of *Oïdium albicans*, which also give to it special microscopical characters. Sordes occur in patches of unequal thickness, very little coherence, and no great extent, and are removeable without injury to the subjacent tissue, however red and tender it may be; they chiefly accumulate in front of the arch of the palate, acquire an acid reaction, and then also become the seat of the *Oïdium albicans.* It is only when the diphtheritic exudation approaches to these local and chemical conditions that it becomes the seat of similar parasitic growths.

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[1] Consultationes, p. 136.

[2] Op. cit. 2d edition, pp. 32, 33. It is not until the publication of the fifth edition, twenty years after this, that an error in the alteration of this foot-note, and an added paragraph to the preface, led to the confusion that has since existed between Fothergill's sore throat and scarlet fever.

[3] British Medical Journal, 1858, p. 173.

Pathology.—The general disease and the local lesions that arise during its continuance have an interdependence and mutual reaction. The latter are not confined to one period of the disease only; they occur throughout its course, sometimes proving fatal by the vital importance of their site, at others adding to its force, and prolonging its continuance. The general disease impresses a special character, not only upon the local lesions which it occasions, but upon any concomitant morbid action: it is marked throughout by an elevation of the normal temperature of the body, by enlargement of the lymphatic glands and the spleen, by a varying amount of congestive action of the liver and kidneys, and of various parts of the mucous surfaces, as well as of that of the fauces and first air-passages, where the speciality of the diseased action is most marked, and where it sometimes expends its whole violence.

The local lesion peculiar to Diphtheria is most readily induced in surfaces exposed to the free access of air, and though not restricted to them it is there that it assumes its most characteristic development; there is a stasis of blood in the capillaries, a destruction of the red corpuscles, and a formation of fibrin, as shown by the spontaneous coagulation of part of the exudation. These changes take place in close contact with the blood-vessels, and commence in the mucous membrane beneath the epithelium, transforming the cells of the subepithelial layer or of the epithelium itself, or altogether replacing them by the fibrinous exudation or false membrane. The false membrane cannot be detached without leaving a bleeding surface, which is rapidly covered with a new layer: it neither assists the cicatrization of the surface on which it forms, nor ever becomes itself organized. Superficial ulceration results from the interstitial necrosis of some parts of the tissue invaded by it, but there is no gangrene or mortification of its substance. Changes of decomposition rapidly take place in the deposit itself, with injurious consequences, both to the lymphatic glands near, and to the system at large, from the absorption of effete matters; the site of these changes may occasion other ill effects, by contaminating the air of respiration on its passage to the lungs. The disappearance of the false membrane is effected partly by this superficial destruction, and partly by absorption from its under surface and edges, and by return to healthy action in the vessels below. It is seldom entirely separated in this way; a thin layer is often left, through which pink points gradually appear; soon only isolated spots of exudation remain, which are finally removed by absorption, gradually effected as the process of reparation proceeds. This is sometimes much retarded by the constitutional debility induced by the disease; and is sometimes interfered with by a retrograde process of ulceration.

The extension of false membrane proceeds, primarily, from the wide dissemination of original centres of its deposit; secondarily, from the invasion of contiguous surfaces; the morbid action may thus extend gradually in every direction, or advance with great rapidity on the parts irritated by the accompanying serous exudation. This may be one cause of implication of the air tubes,[1] and of that progress of the disease from above downwards which Louis was induced to consider as almost the law of its extension. An illustration of the opposite mode of extension is sometimes seen when Diphtheria extends from the nose to the lachrymal duct, and thence to the conjunctiva; or in its progress from the lower edge of the palatine arch to the uvula; the turgescence preceding the exudation causes the uvula to be recurved upon the side already affected; soon the false membrane has not only invested the whole uvula, but extends upwards behind the velum. Wherever false membrane is formed, some degree of inflammatory action is excited; this may either approach very nearly, both in its products and symptoms, to the type of ordinary inflammation, or be entirely subordinated to the influence of the general disease, in which case this peculiarity is remarked—that the less marked the inflammatory condition, the more extensive is the pseudo-membranous formation.

The evidence of the general disease next in importance is the existence of albumen in the urine. Dr. Copland, in his Dictionary, first mentions "albuminous urine" in his account of the pathology of croup, which is framed to include the croupal complications of this disease. We are indebted to Dr. Wade, of Birmingham, for demonstrating the dependence of this system upon Diphtheria. Bouchut and Empis[2] soon after called attention to its importance, and Dr. Sanderson[3] considerably advanced our knowledge of its relation to the general course of the disease, by showing that the presence of a considerable amount of albumen did not interfere with the large excretion of urea which accompanies the progress of the general disease. I possess notes of three cases confirmatory of his observation; in that of J. B. before referred to, thirty-one determinations of the quantities of urea and albumen were made from the sixth to the thirty-seventh day of the disease, by Mr. W. Dunnett Spanton, now of the North Staffordshire Infirmary, at that time (1861) residing with me. On the tenth day of the disease, when the albumen was estimated at one-third, the quantity of urea was twice as much as is normally excreted, the specific gravity being 1016; it was not until the thirty-seventh day that the urea fell to its normal quantity, and albumen was then for the first time absent; the specific gravity had fallen from 1015 to 1010. Subsequent observations on the forty-seventh, fifty-fourth, and sixty-second days, agreed very closely with the last result. The albuminuria is not to be considered as solely dependent on an original change in the blood, but chiefly upon a morbid process in the kidney, which is one of the disseminated lesions of structure occasioned by the general disease. Congestion of the Malpighian tufts is an early lesion, followed by further change in the tubular structure of the kidney; a relation is found between these changes and the amount of albumen, but no constant relation between the albumen and the amount of blood change. In the case of Walker, where albumen did not occur till late in the disease, the blood change was extreme, as evidenced by hæmorrhagic oozing from the palate, petechiæ, and purpura; there was no hæmaturia at any period, and recent disease of the kidney was found *post mortem.* That the function of the kidney may be seriously interfered with, and even suppressed, is shown by a case mentioned by Dr. Gull,[1] and by one reported at the Pathological Society, February 1865, by Dr. Greenhow.[2] Dr. Humphry gives one instance of the occurrence of anasarca,[3] but these cases are rare: albuminuria does not persist after convalescence. Blood changes may be concerned in these symptoms; that these changes are considerable during the progress of the disease is shown by the hæmorrhagic tendency, by the occurrence of fibrinous coagula in the heart and great vessels after death, by the remarkable pallor during the illness, and the anæmia of convalescence, while the frequent affection of the spleen would point to a cause of this aglobulosis of the blood. Whether the lesions of innervation are owing to this cause, to a failure of nutrition in the nerve textures, or to a more special effect of the diphtheritic poison, is doubtful; from the instability of the disordered innervation, and the variety of conditions which it assumes, we may conclude with M. Trousseau, "that the lesion of the nervous centres is not of a very grave character." The impairment of vision is generally, as remarked by Mr. Dixon,[4] due to loss of adjusting power, and there is inaction of the ciliary muscles; he has not found any important change in the retina. Some of the more serious and persistent muscular paralyses are owing to wasting or degeneration of the muscular tissue itself. There are no good reasons for supposing that either special deterioration of the blood, or alteration of its quality, precedes the development of Diphtheria; a state of hyperinosis, if it were possible, is not that induced by many of the diseases to which Diphtheria readily succeeds; the alterations in the properties of the blood, physical, vital, or chemical, are rather the consequences than the cause of the disease; and it would seem that the first influence of the disease, as well as its later effects, are exerted upon the nervous system.

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[1] Not only may noxious matters thus reach the trachea, but detached shreds of secretion from it may be drawn into the smaller bronchi, and mechanically block up, by a kind of embolism, some lobules of the lung, as pointed out by Sir W. Jenner.

L'Union Médicale, No. 132, 1858.

Brit. and For. Med.-Chirurg. Review, January 1860, p. 196.

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[1] Second Report of the Medical Officer of the Privy Council, p. 304.
[2] Transactions of the Pathological Society of London, vol. xvi. p. 47.
[3] British Medical Journal, July 1863, p. 4.
[4] Holmes, System of Surgery, vol. ii. Diseases of the Eye, p. 766.

Morbid Anatomy.—The special product of Diphtheria has affinities with some deposits formed in other diseases, with the products of some forms of inflammation, and with the buffy coat of the blood, which it often closely resembles in appearance and some of its physical properties. It has an alkaline reaction, swells, and becomes transparent in strong acetic acid, and is disintegrated or dissolved by caustic alkalies. It is unaltered by maceration in water, and yields no gelatine to it, as tested by tannin; in this respect it differs from the buffy coat, and from coagulable lymph, and also in giving no evidence of albumen on being boiled; it is stained brown by tincture of iodine, and assumes entirely the character of pure fibrin. It is possible to obtain solution of some specimens in the same way that Denis[1] dissolved recent fibrin, and to find them unmixed with other protein matters; other specimens less fibrillated, or less recent, are not acted upon in this way. The tubes of exudation found in the bronchi will often lose their coherence on maceration in water only, and afford evidence of albumen on boiling, which will not always be the case with the denser membrane from the trachea.

Microscopically the superficial part of the exudation is made up of the epithelium of the membrane on which it occurs, entangled in the upper layers of a transparent homogeneous substance, throughout which are found some altered epithelium cells, granular corpuscles, and nucleolar bodies, in varying proportion; they become less numerous in the lower layer of the exudation, in which blood-corpuscles and pus cells frequently occur. Fibrillation, similar to that of other fibrinous exudations, is sometimes observed on the under-surface of the diphtheritic false membrane, from the fauces. The false membrane from the trachea is corpuscular throughout, as is also the pulpy and granular deposit which is sometimes found in the fauces, and is more frequent in the larynx; these deposits rapidly pass into granular degeneration. In the bronchial exudation the corpuscular element differs little from that observed in the product of ordinary inflammation there; sometimes little flakes of fibrin are found attached to the bronchial membrane, but the only other evidence of its presence is the coherence of the bronchial casts. The laminated, or fibrillar, the pultaceous and the granular, or corpuscular, are the leading forms of this exudation; they may present themselves in any combination under the same morbid influence; in the trachea they are always associated, and either may predominate; the fibrillated may be deposited in the mucous and submucous tissue of the larynx,[2] and the granular may be seen in the fauces together with the laminated, the less coherent deposit, misnominated croupous, being as truly diphtheritic as the more tough, leather-like formation. The one point common to both is their intimate relation to the structure of the membrane on which they are formed. In the case of Bowra I saw deposit in the contiguous edges of the true and false vocal cords, the sacculus laryngis being free, and the commencing granular deposit in the trachea firmly imbedded in the mucous membrane. Dr. Wilson Fox has demonstrated, in all stages of tracheal exudations, lesions of the membrane sometimes exposing the fibrous tissue beneath, both in adults and in children. M. Hache observes[1] the resistance to the separation of the false membrane from the trachea at a certain stage, and calls attention[2] to the sanguineous points found imbedded in its under-surface when removed.

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[1] Arch. Gén. de Méd. s. 3, tom. i. p. 171. Half a drachm of nitre to an ounce of water will effect this at a mean temperature. The solution is imperfect, viscid, coagulates in flakes at 163° Fahr., and is strongly precipitated by acetic acid.

[2] Dr. Bristowe, Trans. Path. Soc. Lond. vol. x. p. 323.

The condition of the tissue in which the morbid action is exerted, though of less importance than the general course of the disease, affords better means of characterising it, than the exudation to which it gives rise; the unsatisfactory nature of a distinction founded upon the two forms of exudation in this disease is thus admitted by the most advanced pathologists of Germany:[3]—"The difference which I formerly established between the croupous and diphtheritic forms, is often lost in particular cases, so that the true croup of the larynx and trachea invades the tissue of the mucous membrane, ay, very often co-exists with Diphtherite at the back of the pharynx and of the fauces; and not till the deeper bronchi are reached, or the pulmonary cells, does it become a free exudation. This is found not only in the epidemic croup of children, but also in Diphtherite occurring in typhoid conditions, and in hospital gangrene; or simultaneously with diphtheritic necrosis of the vaginal and intestinal mucous surfaces." This interstitial necrosis, or ulceration of the surfaces in relation with the exudation, is the anatomical character on which the distinction between the diseases here mentioned and Croup, in our acceptation of the term, must be based.

The diphtheritic deposit may occur in isolated patches, or extend continuously from the nares to the bronchi; it may be moulded on to any anfractuosity of the nasal passages; it may line the whole pharyngeal cavity, adhere to both surfaces of the epiglottis, cover the interior of the larynx, form a cylindrical cast of the trachea, and may thus be removable from even the smaller bronchi. More rarely it is found to extend in this manner throughout the œsophagus; in one of the two cases reported by Bretonneau,[4] it was strongly adherent to the upper part of the tube. In two of seventy-four cases, tabulated in the British Medical Journal,[5] the œsophagus was partially affected, one with, one without extension to the air-passages. In a case reported by Dr. Morley Harrison, of Manchester,[6] the false membrane extended forwards to the mouth, and a patch existed upon the frænum linguæ. Dr. Bristowe gives an instance of the upper part of the œsophagus being covered. Virchow alludes to one case; he has seen the occurrence of false membrane in the gall-bladder.[1]

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[1] Barthez and Rilliet, tome i. p. 318.

[2] Ibid. p. 319.

[3] Virchow, Path. und Therapie, vol. i. p. 292.

[4] These cases, and two others occurring in children with implication of the larynx, are mentioned by Barthez and Rilliet, tome i. p. 322.

[5] Brit. Med. Journ. 1859, pp. 305–6.

[6] Ibid. 1863, vol. i. p. 306. A complete cast of the tube was ejected.

The tonsils are frequently the seat of this formation; it is often detached early, but may accumulate to a surprising thickness, the outer surface retaining for a time the exact appearance of the enlarged tonsils, and marked with the opening of its follicles. I have met with one specimen, which it was difficult to believe was not the organ itself under examination; every part of it was soluble in Denis' solution, and no trace of organized tissue occurred throughout its substance; this exudation has attained to the thickness of two-thirds of an inch.[2] The decomposition of such concrete deposits, their varied colour from admixture of altered blood, and their detachment in offensive shreds, has often given a false idea of the gangrene of the tissue beneath, which really remains almost intact. Bretonneau called attention to the importance of this fact, but while he correctly described the "ecchymoses of no great extent, as well as the slight erosion of the surfaces on which the disease had existed longest,"[3] insisted too much upon the integrity of the mucous membrane being always preserved; but though much may be repaired before death, it is rare not to find some evidence of lesion of the mucous membrane. Sometimes the submucous layer is bare and granular, without defined ulceration;[4] at other times a defined ulcer exists, exposing the muscular fibres.[5] M. Louis noticed erosion of cartilage at the posterior nares. The uvula and part of the soft palate have been lost by ulceration.[6] Dr. Sanderson[7] mentions the division of the left half of the soft palate by a penetrating ulcer. Mr. Simon[8] describes a circular ulcer of the pharynx and other signs of ulceration. Dr. Ashley[9] observed phagedæna commence at one tonsil and extend to the carotid. Ulcerations on either surface of the epiglottis, and on the mucous membrane of the larynx and trachea, are frequently met with. The submucous tissue may be infiltrated with blood, with serum, or with inflammatory products. Dr. Newman[10] reports the formation of an abscess in the palate soon after the disappearance of the superficial exudation. In the case described by Sir T. Watson,[11] pus was found in one tonsil, and this has since been not infrequently observed. Mr. Pound,[12] of Odiham, reports a case of abscess in the pharynx, and Dr. Greenhow[13] gives one of post-pharyngeal abscess. Suppuration rarely, if ever, occurs in the cervical glands; they are large and red, or paler, and brittle, and sometimes present a spleniform disorganization; the swelling around them is from infiltration of serum in the cellular tissue. Dr. Bristowe[1] reports a remarkable effusion of blood among all the tissues of the neck. Various forms of petechiæ, purpura and superficial eschars are found upon the skin, and spots of purpura, petechiæ, and ecchymotic staining are frequent in other parts of the body, as in the muscular tissue and under the serous membranes, as on the lung, upon or within the heart and the peritoneum. I have met with no instance of inflammation of the serous membrane. Fatty degeneration of the muscular tissue of the heart occurred in the case reported by Dr. Bristowe.

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[1] Op. cit. vol. i. p. 292.
[2] Memoirs on Diphtheria. New Sydenham Society, 1859, p. 98.
[3] Traité de la Diphthérite, p. 33.
[4] Dr. J. R. Hughes, Brit. Med. Journal, 1859, p. 80.
[5] Barthez et Rilliet, tome i. pp. 259 and 287.
[6] Greenhow, op. cit. p. 201.
[7] Loc. cit. p. 191.
[8] Trans. Path. Soc. Lond. vol. x. p. 317.
[9] Brit. Med. Journ. 1859, p. 480, case 60.
[10] Ibid. 1863, vol. i. p. 215.
[11] Lectures on the Practice of Physic, vol. i. p. 865. 4th edit.
[12] Brit. Med. Journal, 1858, p. 750. Report of Reading Path. Soc.
[13] Op. cit. p. 237.

Fibrinous coagula are very frequently met with in the cavities of the heart, extending into the great vessels. This occurrence is not limited to cases where there has been obstruction of the air-passages, though they are at least as frequent in the right cavities as in the left. Dr. Barry,[2] of Tunbridge Wells, reports three cases, all in the right side of the heart; the coagulum existed in the right auricle only, in one case, and in another extended into the pulmonary artery. Dr. Rollo[3] gives the case of a soldier where, with the characteristic exudation of Diphtheria, fibrinous polypi were found in the right ventricle. The lungs are frequently congested, and the seat of lobular hepatization in various stages, often sufficiently extensive to be the cause of death, even where no exudation has occurred in the air-passages. Pulmonary apoplexy is sometimes found. Where the air-passages are the seat of the exudation, lobar and lobular pneumonia, the latter often secondary to embolism of the smaller bronchi, with collapse of lung tissue, and acute vesicular emphysema, have generally been induced. The vessels of the brain or of the pia mater have presented fine injection in some cases; in others there has been fulness of the sinuses, and even transudation of blood; and Dr. Gull reports[4] suppurative inflammation of the membranes of the brain and cord, with soft, purulent lymph in the sub-arachnoid space. Dr. Humphry[5] has met with a small spot of suppuration on the under-surface of the left cerebral hemisphere, with softening of the adjacent brain substance, attributable to Diphtheria. The liver is frequently found full, sometimes greatly congested, but is seldom the seat of disease. The stomach often presents serious changes, its mucous membrane being softened, unequally thickened, and red from small patches of deep-coloured congestion, or from extravasated blood; sometimes these changes are found in connexion with isolated patches of opaque deposit at its cardiac end, and the œsophagus presents abrasions of its mucous membrane when it has been the seat of deposit. Vascular injection and minute ecchymoses have been found in the small intestines, the

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Trans. Path. Soc. of London, vol. x. p. 328.

[2] British Medical Journal, 1858, p. 623.

[3] Essay on Cynanche Trachealis, by John Cheyne, M.D. 4to. Edin. 1801. P. 68.

[4] Loc. cit. p. 299.

[5] Brit. Med. Journal, July 1863, p. 4.

follicular orifices are often conspicuous, more rarely Peyer's patches in the lower part of the ileum are very distinct and prominent; sometimes deposit and ulceration occur in the large intestines, hardly distinguishable from that of dysentery. The spleen is generally found full and soft, more frequently paler in colour than redder, and often presenting a cloudy or opaque appearance on section.

The kidneys may often appear healthy to the naked eye, but are very rarely found so under microscopical investigation; they generally present well-marked, and sometimes extreme evidence of special change: this is chiefly in the intra-tubular structure, and though congestion of the Malpighian tufts is often very conspicuous, the general aspect, sometimes mottled from an irregular blending of anæmia and congestion, is more often of a pale than of a deep colour; they have not been found in the extreme state of disorganization sometimes seen after scarlet fever. The tubules appear opaque, from the epithelial cells being numerous, easily detached, and filled with an unusual amount of finely-granular material, in which oily globules are sometimes abundant. Some tubes are devoid of epithelium, and present transparent, fibrinous casts, or these are found separated from the tubes to which they correspond in diameter; occasionally blood-corpuscles are found in the tubules; more frequently they are filled with masses of epithelial cells and fibrinous exudation. These changes are as noticeable in the medullary as in the cortical structure; granular exudation is also found between the Malpighian capillaries and their capsule. The supra-renal capsules have been found intensely vascular.

PROGNOSIS.—No case of Diphtheria is to be regarded without anxiety; every danger incident to the disease may result, though the early symptoms are but slightly marked. The successive appearance of fresh patches of deposit, of albuminuria, or of other signs of the disease, excites alarm, lest its next local manifestation should be in the larynx: the simultaneous occurrence of many of these signs, and the increase of any of them, as of the enlargement of the lymphatic glands, and of the amount of exudation, indicate an intensity of the general disease which the young or the enfeebled will hardly withstand, and which, with implication of the larynx, will be rapidly fatal. The least laryngeal quality of the voice or cough, and especially of the respiration, is a sign of the greatest danger; it may, in the strongest person, soon end in fatal obstruction of the glottis from the occurrence of exudation there, or it may be the first indication of the equally fatal and more insidious extension of it to the bronchi. Much exudation in the nasal passage is unfavourable. If both nares are occluded, respiration must be carried on by the mouth with great discomfort; this may be a cause of death in young children, as they are thus unable to suck. The occurrence of hæmorrhages is a bad and dangerous symptom; so is repeated vomiting and purging at the commencement of the attack. These symptoms occurring somewhat later, bring on death by exhaustion, and indicate either serious alteration in the blood, or that the stomach has become the seat of the disease. A very rapid pulse, except at the commencement, is indicative of danger; so also if the pulse fall below the normal frequency. Albuminuria, if unattended by any rise in temperature, is in itself not serious as long as the urine is in normal quantity, without blood corpuscles or casts of tubes, and while the specific gravity continues to be high. Any increase in the temperature of the body after the first five days, or a persistence of high temperature after the first ten days, is unfavourable; a sudden rise may indicate danger from some intercurrent disease. There is also the danger of relapses from the slightest debilitating causes, either in the earliest or at the latest periods of the illness. The liability to laryngeal obstruction is greatest towards the end of the first week; it begins with the commencement of the disease, and probably continues throughout; this complication has happened as late as the fourteenth day,[1] and possibly later.[2] These dangers are especially treacherous, as they may occur in cases where there has been but little exudation on the pharynx, and as a period of comparative convalescence may intervene between either the first symptoms and the laryngeal complication, or between those of the more advanced disease and its asthenic accidents. The first impression of the general disease may be so profound as to make recovery impossible; on the other hand, cases commencing with marked constitutional reaction, and with symptoms generally unfavourable, such as vomiting, epistaxis, or nocturnal delirium, may recover without experiencing the more serious consequences of the disease.

Certain epidemics and certain periods of them are marked by a great fatality, and instances have occurred in which all or nearly all of those seized have died. Age and family constitution have the greatest influence; where one member of a family has suffered severely from this disease, there is the more reason to dread its effects upon the younger members of the same family. The proportion of deaths to seizures in 1,321 cases reported in the *British Medical Journal* for 1858-59, is one in seven; among the severer cases, and at the height of any local epidemic outbreak, it was as high as one in three; in other cases it was less than one in ten. Of the seventy-four cases collected by the *British Medical Journal*,[3] twenty-six died,—fourteen from asthenia, eight from implication of the larynx, three from syncope,

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[1] Etude de la Diphthérite, par G. S. Empis. Arch. Gén. de Méd. 1850, s. 4, tome xxii. p. 298.

[2] Cases of Diphtheria, by G. M. Humphry, M.D. Case 7. Brit. Med. Journal, July 4, 1863. Dr. Ballard tabulates one case, loc. cit. p. 55.

[3] Brit. Med. Journal, 1859, p. 498. Of eighty fatal cases occurring in Islington in 1858-59, Dr. Ballard found that in fifty-eight deaths twenty-seven were from laryngeal affection, and twenty-three from exhaustion; thus distributed as to age and period of illness :—

	Under 5 Years.	Under 10 Years.	10 Years & upwards.	1st Week.	2d Week.	3d Week.	4th Week.
Laryngeal affection .	17	7	3	18	8	1	...
Exhaustion	13	5	5	8	8	3	4

and one from subsequent bronchitis. These cases were mostly severe, and seldom under treatment at their commencement. One of the most important conditions of a favourable result is the early recognition and treatment of the disease.

THERAPEUTICS.—In Diphtheria both local and general means of treatment are required; the cure of particular cases may sometimes be attributable to the one, sometimes to the other, but in no case can either be safely disregarded. The general therapeutical indications are of primary importance throughout; they consist neither in attempts to nullify a poison by specifics, nor to expel it by elimination, but in withstanding the encroachment of the disease, and in sustaining the vital powers.

Complete rest and purity of air are essential. Alcoholic stimulants are required throughout; they are as often as serviceable early in the disease as in that part of its course when they become indispensable. A rapid pulse indicates their employment, and heat of skin is no counter-indication. The limit to their administration should be calculated according to the age of the patient and the amount of bland liquid representing water that can be taken at the same time. In certain cases one or two full doses of quinine may be given, often with good effect when there is either vertigo, headache, or vomiting; soon afterwards beef-tea, eggs, or even more solid food, can be taken, as well as the brandy or wine; milk, in any form, is always suitable. The night must not pass without either nourishment or stimulant being given; wakefulness or nocturnal delirium is often thus obviated; sometimes a dose of morphia or opium has to be combined with the stimulant, after the second or third night, if restlessness then persist.

The condition of the fauces must be alleviated by the free use of ice sucked or swallowed, and by any of the soothing means hereafter mentioned internally, and by moderately warm applications externally. When the patient first comes under observation, a patch of exudation may be apparent, such as to require energetic topical measures. For this purpose a solution of nitrate of silver should be applied so as thoroughly to come into contact both with the patch and the turgid mucous membrane surrounding it. The strength of the solution should not exceed the proportion of one part of nitrate of silver to three of distilled water; the superficial whiteness left by it will clear off in twenty-four hours, and is easily distinguishable from the points of exudation. A mixture of hydrochloric acid and honey in equal proportions, or with one or two parts of water, is as effectual in checking the progress of the exudation, but leaves a more persistent white mark. These applications, when seen to be efficient, need not be repeated; care is to be taken that no excess of them reach beyond where they are required. The strong acid and solid nitrate of silver are both objectionable.[1] Where there is much redness and pain a weaker solution of nitrate of silver, one part to eight or twelve of water, pencilled over the whole surface, tends to prevent further exudation, and affords relief to the local discomfort. Hydrochloric acid, diluted with five parts of water, may be applied in the same way with the same effect.

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[1] Extreme dysphagia has at once subsided on the use of solid nitrate to a foul or irritable ulcer consequent upon Diphtheria.

As soon as nourishment, however light, can be retained by the stomach, five to ten grains of the perchloride of iron, the equivalent of twenty or forty minims of the tincture, should be given, with not less than half an ounce of water and half a drachm of glycerine; this should be repeated every three or four hours, or still more frequently, so that as much as a drachm of the iron, or half an ounce of the tincture, be taken in the course of twenty-four hours. This quantity may be reached even in children, during severe attacks. It should be commenced on the first day of the illness, or as soon as the patient comes under notice, and continued till the tongue becomes red and the throat improves; when deposit has already taken place, the good effect of the remedy will be shown, not by any alteration in the dimensions of the patch, but by a diminution in the accompanying secretion, and by an improvement in the general symptoms; it is not to be discontinued for some days, and may require energetic repetition if improvement is slow in appearing. Dr. Druitt[1] reports a case where great benefit followed upon the quantity of the tincture of the muriate of iron, administered under the direction of one of the physicians of the Middlesex Hospital, amounting to one ounce and a half in the twelve hours. Mr. Hamilton Bell,[2] of Edinburgh, first proved the utility of iron in this form and quantity in erysipelas, and his brother, Dr. C. Bell, advocated its employment in analogous diseases; hence its application in Diphtheria. Dr. Godfrey,[3] of Enfield, used it in this way, and its use was further recommended by Dr. Heslop, of Birmingham; it was employed with advantage in the Boulogne epidemic, and M. Aubrun[4] gives the result of three years' favourable experience. The solutions of the pernitrate of iron, or of the acetate, answer equally well when given in proportionate doses, the latter being stronger, the former of less strength, than the tincture of the perchloride. A local as well as a general influence is exerted by these agents; they have a constringing effect on the vessels, and their action on the decomposing exudation is antiseptic; their general effect is as much owing to a topical action on the whole gastro-intestinal canal, as to absorption, for when the prescribed quantity of the persalt of iron is reduced by soda in the presence of citric acid, and so administered in a soluble form little liable to decomposition, and readily available for absorption, no favourable effect is produced; indeed, none of the protosalts of iron are equally efficacious at the outset of the disease.

The local action upon the surfaces affected is certainly important;

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[1] Brit. Med. Journ. 1861, vol. i. p. 208.

[2] Edin. Monthly Journal, June 1851.

[3] Lancet, October 17, 1857.

[4] Gazette Méd. de Paris, December 8, 1860, f. 761.

and when the nasal passages are implicated, syringing them with the perchloride in a solution slightly weaker than that prescribed for a draught, is essential; half a drachm each of the tincture of the perchloride of iron and glycerine with six or seven drachms of water, may be injected into the nares by an ordinary glass syringe, the point of which is shielded by a piece of india-rubber tubing, or into the upper part of the pharynx by means of the laryngeal syringe directed upwards. These parts may be gently and efficiently irrigated by placing a vessel of any fit liquid at an elevation, and inserting an india-rubber tube from it into either nostril. When the patch is already formed, and the secretion in the fauces considerable, it is well to apply a stronger solution of the perchloride, not exceeding the strength of the tincture, by means of a full-sized camel's hair-brush, both to the patch and to the adjacent surfaces. A patch of considerable thickness, and intimately adherent to the membrane, may be touched with a preparation twice the strength of the tincture, made by mixing equal parts of the liquor ferri perchloridi and of glycerine; this application is strongly styptic, and should be confined to the surface of the exudation, which it readily penetrates: and exerts its influence upon the vessels beneath. Where the use of the persalts of iron is commenced early, and persisted in, the necessity for local application to the throat is often obviated, and it is better to encounter a little difficulty in the administration of the medicine, which may be given in any degree of dilution, than to be obliged to resort to the always disagreeable, though often indispensable, performance of topical medication. The medicine is better given at the time of taking nourishment, the pain of swallowing diminishes under its use, and its application in any way to the whole pharynx affords relief.

Difficulty of deglutition is often a serious aggravation of the illness. Much benefit is derived from the application of the perchloride of iron diluted with water to the strength of the tincture, but with a further addition of glycerine, to the pharynx by means of a camel's hair-brush two or three times in the first twenty-four hours. Pure glycerine[1] applied in this way is very soothing, and the frequent use of more simple means conduces to relief. A lotion of acetate of lead, gr. ij. ad ℥j., relieves. A weak solution of tannin, with a little chlorinated soda, is useful. Lime water as a wash, or gargle if the effort be not too painful, alleviates; so does the injection of cold water into the pharynx. The most grateful, and in all cases one of the most useful appliances, is ice in small pieces, dissolving in the mouth. Weak solutions of borax, or of alum, with honey or glycerine, or a strong solution of chlorate of potash, are useful where there is much tenacious secretion; the latter salt in coarse powder, or small crystals of it, may be taken into the mouth from time to time with benefit. Any of these may be used either cold or tepid as may be most grateful to the patient, and when there is fœtor, or the glands are much swollen,

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[1] Dr. Mayer, American Journal of Med. Science, April 1858.

Condy's fluid (permanganate of potash), largely diluted, or chlorinated soda, one part of the strong solution to twelve or fifteen parts of water, is to be used so as to wash away any detached exudation, and this by means of a syringe if not otherwise easily manageable. The strong solution of chlorinated soda may be directly applied to any foul surface not yet detached, or to any foul ulceration when there is induration of the external glands. The Pharmacopœial glycerine of carbolic acid is a convenient local remedy when decomposing matters are tainting the breath. The similar glycerine of gallic acid checks hæmorrhage from parts whence the slough is separating. The glycerine of tannin is most useful in the earlier stages of the disease, and is a necessary application to the fauces where bark with a mineral acid in the place of iron and quinine is the general medication adopted. Semi-detached shreds of decomposing matter are to be carefully removed; such matters cannot be allowed to remain with safety where they are; they must be reduced to the smallest amount possible, and antiseptics employed until they disappear. When exudation ceases, the necessity for local application has passed; some portions of deposit will be slow in clearing away, and may remain without interference; abrasions or ulcerations of the mucous membrane heal most quickly when left alone. A tender surface is never to be exposed by the forcible removal of any adherent exudation.[1]

All the remedies of special utility in Diphtheria are antiseptic: the stronger forms above indicated are limited in their application to parts that can be brought within the range of sight or touch. This range may in some states of the disease be extended by the use of the laryngoscope not only in the direction of the larynx, but also towards the posterior nares.

Many of these agents can be most advantageously used in all stages of the treatment by atomizing their dilute aqueous solutions in the manner first suggested by M. de Sales Giron. Any of the spray-producers now in use will effect this, and supply a ready means of diffusing the remedies over every part of the mucous surfaces obnoxious to the disease. Dr. Dewar, of Kirkcaldy, has shown that the strong sulphurous acid of the Pharmacopœia may be used at short intervals with benefit, and has devised a portable instrument with a vulcanite tube for the purpose. In one case where this was used without resort to the iron treatment in a child, no laryngeal extension of the disease occurred. A mixture of sulphurous acid and honey for frequent deglutition was useful in clearing the posterior nares; the fauces required an occasional application of glycerine of tannin; on the fourteenth day acute kidney complication began, and terminated fatally on the eighteenth day. When the tincture of perchloride of iron is given (generally combined with quinine), very little local treatment other than the spray is resorted to. Besides the weak Condy's fluid, a still weaker solution of carbolic acid, a teaspoonful to a pint of water, may be used in this way; a few spoonfuls of the latter solution is advantageously given occasionally as a drink. Both these liquids should be kept in the sick-room in a stronger from, both for diffusion into the air of the room by means of the vaporizer, and ready for disinfecting any secretions or soiled clothes.

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[1] Ætii Amid. tetrab. 2, serm. 8, cap. 46: "Nam inscii ad quos in rebus dubiis præcipuè homines confugiunt, vehementius illiniunt, simùlque inflammatum locum comprimunt, simùlque crustam detrahunt; quod minimè facere convenit, priusquam elevatam et vix innitentem crustam conspiciamus; quod si enim adhærentem adhuc crustam avellere aggrediamur ulcerationes magis in profundum procedunt et inflammationes consequuntur, augentur dolores, et in ulcera serpentia proficiunt: itaque sicca remedia insufflare oportet, liquida vero cum pinnula illinito, ita ut quantum licuerit, pinnulam penitissimè immittamus." "Ex quo præcepto docent medici, quod cùm pueri nequeunt gargarismatis uti, injiciantur cum siringâ medicamenta liquida." (Heredia op. Med. tom. iii. sec. 3, cap. xii.)

There are other remedies that sometimes are necessary in checking the spread of Diphtherite, or in modifying its character. The salts of copper, as used by Aretæus, are very effective, but their use is not free from danger. Alum in powder (the *poudre croupale* of Pommier) is as effective, and both safe and simple in use; it may either be used alone, or mixed with one-third of its bulk of finely-powdered nitrate or chlorate of potash, or with a less proportion of sugar or gum; it may be applied to the throat by insufflation, as practised by Bretonneau,[1] or by making it into a paste with a drop of water, and carrying it to the part by means of a rod or spatula, or on the handle of a tablespoon. When required in the upper or lower part of the pharynx, or indeed on any part from the gums to the glottis, that best and safest of all instruments, the end of the finger, may be used for this purpose; in the same way, with the protection of a little cotton wool wrapped around it, the other local remedies can be carried to any spot determined upon, without at the moment requiring the aid of sight. Calomel has an undoubtedly useful topical effect, as is seen in its application to cutaneous Diphtherite. Any sore on the skin should receive careful attention, as, however trivial at first, it may seriously compromise the prospect of recovery, either by spreading or by deepening so as to lay bare the subjacent structures. In addition to the requisite escharotics, cotton wool should be used as a dressing; greasy applications are injurious.

The care given to the local treatment will fail of success, the diseased action checked at one part will reappear, or again extend rapidly, if the general condition necessary for a safe conduct through the illness be disregarded. Children, when improving, must continue in bed; it is the only place where they get rest of limb. There must also be precautions against fatigue from over-amusement or excitement. The period during which rest is to be enjoined is not merely while exudation remains, but while any symptom of the disease continues, such as slightly elevated temperature, excess in the excretion of urea or urates, or albumen in the urine; care is still more necessary in providing against any undue expenditure of strength during some of the after effects of the disease.

As long as solid food cannot be taken, it is to be noted with the greatest exactitude that the quantity of the liquid nourishment and of stimulant administered in the twenty-four hours is equal to the estimated requirement of the patient; the less the quantity of nourishment, the greater must be the dependence upon the stimulant. The youngest children may require a tea-spoonful of brandy every two hours; a child of three years old two tea-spoonfuls. This may be given diluted in any way and in very small quantities, frequently repeated. Older children take it best mixed with iced water or soda-water. Champagne is often a good substitute; port wine requires dilution, except for adults, who also find good claret, red hock, or some of the stronger Hungarian wines, suitable. The quantity of stimulant ordered must be considered in relation not only to the immediate necessity that may exist for its employment, but also to the probable course of the disease and the strength of the patient; a moderate quantity, repeated at regular intervals, is of most service in maintaining the strength of the patient where all the symptoms are well marked and likely to go through their full stages; at certain crises of the disease, or where some symptoms only have attained great prominence, the benefit that results from a bold resort to stimulants is surprising, and the effect is the more marked, if their use had up to that time been neglected. Where the exudation is checked and is first separating, I have known the determined deglutition of four pints of beef-tea and nearly two pints of port wine, in little more than twelve hours, by a temperate young man, teacher in a school, put a stop to further illness. Mr. M'Donald,[1] of Bristol, relates a case where bottled bitter-beer being the stimulant ordered, nearly eight pints were taken (twelve pint bottles were emptied) in the course of one night, with a like good effect. At a further period of the disease, when the separation of the deposit is completed, extra stimulants are required to combat the restlessness and depression then sometimes extreme. Sleep, at all times necessary, is at this latter period of the illness to be carefully conciliated. Opiates are well borne and are now more likely to procure sleep than when the first symptoms in the throat are most troublesome; their use for several nights, when the diminished exudation reveals injury of the mucous membrane, may do much to prevent the exhaustion at this time so dangerous, and upon which stimulants, beyond what is necessary for support, seem to have but little power. Quinine and bark are particularly serviceable; the former may be combined with iron if its use be still indicated, the latter with the mineral acids. Quinine can be given to children in powder, and in solution is often advantageously added to the nutrient enemata that must so frequently supplement the inadequate amount of support otherwise received. Brandy is occasionally to be administered in this way, care being taken not to set up an irritability of bowel that might interfere with the continued employment of a means of sustaining life that so often becomes a necessity in the treatment of this disease.

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[1] Archiv. Gén. de Méd. tome xiii. p. 5, and tome xvii. p. 508.

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[1] Lancet, 1858, vol. ii. p. 539.

Aperients are seldom required at the commencement of the illness, and during the earlier stages their effects are injurious; for then the waste of tissue is most active, and the impediment to the reception of nutrition the greatest. It has been a matter of frequent remark that exudation in the throat, already stationary, or even diminishing, has at once extended after the moderate action of aperient medicine. The only indication for their employment would be occasioned by intercurrent disease, which would give rise to a greater elevation of temperature than usually obtains in uncomplicated Diphtheria; otherwise several days may be allowed to pass without action of the bowels.[1] The appearance of albuminuria does not call for their administration. Mr. Spanton found an increased excretion of urea follow the action of a purgative. Diarrhœa frequently demands attention; opiate injections, or small doses of opium with bismuth, will generally suffice; alum in boluses is mentioned by Bretonneau; small doses of sulphate of copper with opium are best in severe cases. The constipation of the latter stages of the illness requires stimulating enemata, friction to the abdomen, and galvanism.

Salines are contra-indicated. I have seen no good result from the administration of the alkaline remedies recommended by some French authors in the earlier stages, nor from the use of the iodides or bromides in the later. Ammonia, in continued doses, is injurious;[2] as a stimulant it is useless. The diaphoretic action of salines is not required, and their diuretic action has an unfavourable influence upon the specific irritation of the kidney.

Diluents and acid drinks should be continued while albuminuria persists; and though stimulants are not then always to be withdrawn, they are not to be injudiciously augmented. The hot air bath applied to the body only, without removal from bed, or warm packing to the loins, is serviceable in this complication; the appearance of blood-corpuscles or of tube casts in the urine indicates the administration of iron with the mineral acids, and the addition of cod-liver oil.

Mercurials, if continued till their general effect upon the system is produced, are productive of much mischief; Bretonneau's second memoir gives evidence of this. The carefully regulated action of them may be usefully opposed, when desirable, to some of the inflammatory complications of the disease. Salivation rather favours than checks the rapid extension of the special exudation.[3]

Bleeding is specially to be avoided; the worst effects of the disease seem to have been more extensively developed in many of the cases where this was practised.[4] Antimony also is prejudicial.

When the progress of the disease under the influence of appropriate general treatment seems most encouraging, death may suddenly threaten from impeded access of air to the lungs; relief then depends upon energetic local treatment, the same general means not being neglected. Symptoms that, did we not know their cause, would suggest the free employment of bleeding or antimony for their relief, may, if their cause is patent upon inspection of the throat, be efficiently obviated by a styptic application to the extending exudation, or to the orifice of the glottis itself, and their increase or return prevented by the free use of iron and wine. Nor are these to be entirely suspended if the mischief be altogether within the glottis. Where this is the case the symptoms are more gradual in their appearance and may sometimes be alleviated by giving chlorate of potash, four grains to a dessert-spoonful of water, by the insufflation of alum, or by the application of it to either surface of the epiglottis, or of glycerine to the edge of the glottis: the diffusion into the throat of a strong solution of chlorate of potash, or of sulphurous acid, by means of a vaporizer, may also be useful. It will depend upon the type of the general disease whether one or more doses of calomel are to be administered; the laryngeal symptoms once established, the resort to an aperient may be advisable, and calomel is the best that can be employed; where calomel will bear repetition emetics are also admissible. Senega may be used with this object. If ipecacuanha is chosen, ten grains of sulphate of zinc should be added. When secretion is abundant, alum, as already directed,[1] or sulphate of copper (five grains to the ounce of water), given in divided doses (a tea-spoonful only for young children), is the best and most effectual. Emesis is not to be frequently repeated, and it is only when readily induced that it can be otherwise than prejudicial. Real and obvious relief, with increasing fulness of the chest expansion, is not only the encouragement for giving these means a trial, but the sole warrant for trusting to them for the relief of the temporary exacerbations. They must not be trusted to implicitly, as at any moment it may be evident that the obstruction is beyond their influence.

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[1] Nuñes gives a caution against aperients at the latter stages of the illness, lest the death of the patient should be attributed to their use. Heredia, tom. iii. s. 3, cap. xi.

[2] See also Huxham's "Dissertation on the Malignant Ulcerous Sore Throat." Lond. 8vo. 1759, p. 53 et seq.

[3] See Lancet, 1838–39, vol. i. pp. 726, 728, for two cases illustrative of this.

[4] Memoirs on Diphtheria, pp. 50, 97. Arch. Gén. de Méd. tome xvii. pp 494–7.

Tracheotomy should be performed whenever the increasing recession of the softer parts of the thoracic parietes shows that the cause of obstruction to the entrance of air is increasing. In the greater number of cases, if the local medication of the glottis and larynx do not suffice to obviate the danger, tracheotomy, performed early, is much more likely to be successful than after the use of remedies that in any way impair the vital powers. A delay that admits of secretions accumulating in the bronchi is dangerous, and extension of the disease to the lung is the one insurmountable obstacle to success. Where the effects of the obstruction are more suddenly induced, tracheotomy, performed at the very last moment of apparent life, may save it. No degree of severity in the general disease should interfere with this means of averting threatened death from asphyxia, unless the presence of some other complication, necessarily fatal, can be demonstrated. I recently saw a case in consultation with Mr. Adams, in which, had it occurred at the commencement of the epidemic instead of towards the end, I should have decided against tracheotomy, concluding that it must end fatally; although unconsciousness had set in before commencing to operate, the child, six years old, recovered. At some periods of an epidemic, success less frequently attends this operation than at others; at the Hôpital des Enfants Malades,[1] in 1851, there were twelve recoveries in thirty-one operations; in 1853 only seven recovered in sixty-one cases; in 1856 tracheotomy was performed there fifty-four times, with success in fifteen cases.[2] Of 466 operations at this hospital in eight years there were 126 recoveries; these were generally operated upon as soon as the laryngeal symptoms were decided.[3] The result of M. Trousseau's 200 operations gives a similar success of more than one in four, while in the cases operated on by him in private practice one-half have recovered;[4] of nine such cases in 1854 seven were cured.[5] Of forty-two cases reported by Professor Rosen of Tubingen,[6] nineteen recovered. In six of the cases asphyxia had advanced too far before the operation; and of the subsequent deaths, one took place from pneumonia, fifteen days after, and one from albuminuria in the third week.[7] Dr. Buchanan writes, "I have performed tracheotomy twenty-one times, with the result of seven recoveries; and if it be remembered that the patients were all on the point of death from suffocation, it cannot but be regarded as an encouragement to the surgeon to endeavour to save life by operative interference in the later stages of this most fatal disease." After the operation the chief condition of success is efficient support. The tube must remain in the trachea for at least a week; liquids are to be given in the form of sop; and nutrient enemata with brandy will generally be required. A little steam should be diffused in the apartment, and the tube lightly covered with gauze or porous woollen material. In adults, laryngotomy is often sufficient; in one case that occurred to me, the admission of air in this way sufficed to expel the exudation so speedily, that no tube was required.

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[1] *Vide* Treatment of Secondary Croup, p. 104.

The after-treatment of Diphtheria requires great care in proportioning the amount of exertion to the degree of strength existing. Good food, good air, and tonics are necessary. Galvanism may be required when an important function is impeded from muscular inaction.

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[1] Arch. Gén. de Méd. s. 5, tome v. p. 360.

[2] M. André. See art. Croup, p. 101.

[3] Edin. Med. Journal, vol. v. p. 417.

[4] Clinique Médicale. Paris, 1861, p. 414.

[5] Trousseau, De la Trachéotomie dans la Période extrème du Croup, &c. Arch. Gén. de Méd. s. 5, tome v. p. 360.

[6] Sydenham Society's Year-book, 1863, p. 278.

[7] On Tracheotomy in Diphtheria. By George Buchanan, A.M. M.D. Glasgow, 1865.

SCARLET FEVER.

BY SAMUEL JONES GEE, M.D. LOND.

DEFINITION.—An acute pyretic disease, specific in its cause and course, and best characterised by a peculiar exanthem hereafter to be described.

The earliest record which we possess of the existence of Scarlet Fever bears no more ancient date than A.D. 1556, the year wherein Ph. Ingrassias published a description of a malady which had been previously recognised by the common people, and named by them Rossalia. It is possible that examples of a severer type of the same disease formed part of the famous epidemic of malignant angina which raged, sixty years later, in the south of Europe; in Germany, about this time, Sennertus witnessed what he considered to be the rossalia of the Italians, and what we may readily admit to have been our Scarlet Fever. In 1676, appeared Sydenham's short chapter on "Febris Scarlatina," and by the end of that century the disease had been described in every part of civilized Europe.

CAUSES.—I. Contagion is the only known cause of Scarlet Fever. In degree of contagiousness, the disease takes a place between measles and hooping-cough above, and typhus fever below; diphtheria being very far below. The contagious material may be taken up by clothes, and retained by them for a great length of time. Sir Thomas Watson narrates an instance in which a strip of flannel remained contagious for at least a year; and Hildenbrand was infected by a cloak which, after exposure to the disease, had been put aside for eighteen months: hence we may assume that the morbific principle of Scarlet Fever is anything but volatile or unstable. Which being so, affords an answer to the important question: When does a person who has recovered from an attack of the disease cease to be contagious? To speak strictly, not until those natural fomites, the epithelial scales, which were existing at the time of the fever, have been removed; or, what is nearly the same, not until desquamation has ceased. And in the fact that, under ordinary circumstances, these epithelial scales are all but permanently contagious, we have an explanation of the tenacity with which danger clings to materials of any but the closest texture. Uncovering a scarlet fever patient in the direct rays of the sun, a cloud of fine dust may be seen to rise from the body; contagious dust, which, no doubt, subsides into every crevice near the bed. The distance at which the disease may be communicated is commonly said to be not more than a few feet; yet, considering the slight volatility of the poison, one is quite prepared to admit the possibility of what is said to have occurred,—namely, of the contagion having been conveyed hundreds of miles by letter, or similar means. In like manner a person, himself not liable to the disease, may become a travelling disseminator of contagion: a trite observation, the application of which to practice is easily overstrained.

II. Such being the exciting cause, it is required that the subject be apt or predisposed. 1. This aptitude does not exist, as a rule, in those who have passed through one attack of the disease; still the possibility of recurrence and relapse is admitted on all hands. 2. Of persons not protected by a previous attack, whether the liability to contract the disease diminishes with increase of years or not, is not easy to say. The affirmative is probably true, yet no age is exempt. The percentage of deaths at different ages, according to Dr. Richardson, is,—

Under 5.	From 5 to 10.	10 to 20.	20 to 40.	40 and upwards.
67·63	24·43	5·52	1·73	0·66

Indeed all experience goes to prove that the majority of scarlet fever patients are of an age between eighteen months and six years. The relative proportion of cases of Scarlet Fever to all cases of illness, is greatest from four to nine years inclusive, and is, moreover, within those years, nearly stationary. 3. The two sexes are equally liable to Scarlet Fever: women, after puberty, suffer more frequently than men, because more exposed to contagion.

Scarlet Fever appears at any season; but in England especially prevails, as Sydenham says, at the end of summer; or, more strictly speaking, from the middle of September to the middle of November.

Incubation Period.—The duration of the incubation period partakes of that irregularity which attends most of the points which go to make up the history of Scarlet Fever. Trousseau relates a case which seems to prove that the incubation occasionally lasts no longer than twenty-four hours; this we may accept as the minimum. Positively to fix the maximum is impossible: probably seven days are rarely exceeded; but I have myself been witness to facts which did not admit of explanation otherwise than by presuming that the incubation period may sometimes last three weeks. Rilliet and Barthez are of the same opinion.

SYMPTOMS.—Scarlet Fever is sometimes so mild as to escape the observation of both patient and physician, or, again, is sometimes so severe as to kill inevitably within twelve hours: another illustration of the wonted irregularity of the disease. Between these extremes lies a mean or typical form (here first to be described), in which all the characteristic symptoms are well developed, and none excessively: a description of the abnormal forms will follow. In the last place will be noticed the complications and sequelæ which may supervene upon any form of the disease.

ORDINARY SCARLET FEVER.

I. *Invasion Period.*—That stage which precedes the eruption of the rash.

The onset (except in mild cases) is sudden, so that it is often easy to fix the hour, almost the very minute, at which the disease began.

The symptoms are: 1. Sore throat (the first symptoms noticed by most adults), tenderness at the angles of the lower jaw, and stiffness of the neck soon follow. 2. Vomiting, the warning children give, but not so common with adults; it may be repeated many times, becoming ultimately bilious, yet not prognosticate, as a necessity, severity of the ensuing disease: active diarrhœa sometimes concurs. 3. Pyrexia. The first febrile symptom is often, not always, a sensation of chilliness; never a rigor; the face is pale. Flushing of the face and great heat rapidly succeed; a temperature of 104·75° may be reached on the first day. Pulse remarkably frequent, and frequent out of proportion to the height of the fever; for the pulse of a child to reach 160, or even more, on the first day of the disease, in prognosis; signifies nothing; in diagnosis, Trousseau thinks the symptom might be of some value. Respiration in proportion to pulse; no cough; no lacrymation: coryza before the appearance of the rash is very unusual. The tongue, mostly covered with a light white fur, except at the tip and edges, which are red, in some cases remains quite pale, clean, and moist; there are loss of appetite and thirst in marked instances of the disease. Skin hot, not necessarily dry. 4. Nervous symptoms are present, languor, sleepiness by day (especially in children), disturbed sleep at night, nocturnal delirium; headache, not severe, frontal; aching of the limbs. Convulsions even may precede the rash in non-malignant Scarlet Fever; coma, according to Valleix, is a frequent precursor of the rash of the normal disease, and Sydenham makes a similar observation. Lastly, in mild cases, mere poorliness the day before the eruption is often the only premonitory symptom.

The duration of this stage is, as a rule, from twelve to thirty hours. In trivial cases the rash is sometimes the first symptom of the disease. Not rarely the duration seems to be longer than the maximum given above; but proof is difficult; and the difficulty lies in fixing the time of occurrence, not of the first invasion symptom, but of the eruption; sometimes the rash recedes for a short time, and then comes out again. In a case observed by Trousseau, the rash did not appear before the eighth day, upon the cessation of most unusual prodromata, squinting, infrequent pulse and stupor, with headache, and vomiting.

II. *Eruptive Period.*—1. *The Rash.* The normal exanthem consists of small dots, in colour bright scarlet, most intense at the centre of the dot, fading towards the periphery; confluent by their margins, so as not to leave any skin of normal appearance between; not elevated to the touch; completely disappearing under pressure, and rapidly reappearing when the pressure is removed. The rash sometimes comes out over the whole body at once, but is mostly at first limited to a much smaller area, especially to the sides of the neck and the upper part of the chest. Occasionally the eruption appears first on the legs. The most common departures from the type are the following:—The colour may be deep, purplish, dusky (this being, alone, no sign of malignancy): in such a case add numerous flea-bites, and the disease may be (and has been) mistaken for typhus fever. On the other hand, the colour may be very pale. The puncta are sometimes set so closely as to produce a uniform blush, in larger or smaller patches; this condition is constant indeed on the cheeks, and often seen on other parts of the face, and below the knees. Again the puncta may be perfectly discrete. Wherever there is hyperæmia there must be swelling of the cutis, not commonly perceptible, however, simply because the spots are confluent, but when they are discrete, it is not impossible to feel their elevation (sc. papulosa); in the latter case, should the colour be at the same time more purple than usual, the rash of measles will be closely simulated. This swelling of the cutis must not be confounded with other occasional accompaniments of the rash,—namely, a cutis anserina, and a subcutaneous swelling, which is sometimes present to a notable degree in the eyelids, hands, and feet, and which may persist for a day or two after the rash has gone.

The eruption reaches its maximum extent and intensity on the third or fourth day of the illness (occasionally as early as the second); begins to fade on the fourth, fifth, or sixth day; and lasts altogether from five to ten days.

When the nail is firmly drawn over the skin in which the rash is present (over the belly or thigh is best) a white streak soon follows, lasts a minute, and then disappears; a very firm stroke brings out a middle red mark, and two lateral white streaks. These phenomena have been thought to be pathognomonic. But are they really peculiar to Scarlet Fever? Does the "white streak" show more than that the skin is injected, whereby the effect of a contraction of the small vessels in the cutis is heightened, and of a dilatation, obscured?

Miliaria are frequently, but far from constantly present. They are most commonly met with about the sides of the neck, and upper part of the chest, but may be nearly universal. Occasionally they are so thickly set that the epidermis is detached by a rough touch. They are not connected with unusual sweating, but rather with the age of the patient and the amount of eruptive swelling of the cutis; the rash rarely being intense in an adult without the presence of miliaria. They dry up in a day or two and desquamate.

Sc. pemphigoidea seu pustulosa is described by continental writers. Large patches of urticaria sometimes come out while the proper rash is still present.

When the skin is pulled upon, the rash does not always altogether disappear; a yellow stain may be left, or all grades between this condition and actual petechiæ. Like a dusky rash, petechiæ alone do not indicate malignity.

2. *The Sore-throat* is always present to some degree. In a typical case, examination of the throat detects increased redness of the soft palate, uvula, and tonsils, sometimes of the posterior wall of the pharynx, and of the tip of the epiglottis. These parts are swelled also, but the amount of redness and swelling is often disproportionate; the latter may be so great that the tonsils meet in the middle line, and thrust the uvula forwards. Such great swelling is due to causes over and above the hyperæmia, namely, (1) Œdema; this is most notable in the uvula, which from gravitation of the serosity becomes club-shaped. (2) Excess of secretion: this enlarges the tonsils; *post mortem* they are found to be surcharged with thick yellow matter; during life the same secretion often appears at the mouths of the tonsillar crypts, or is exuded so as to cover the surface with a uniform layer. Superficial ulceration of the tonsil sometimes coincides. More rarely the matter does not escape, and, liquefying, causes an abscess in the tonsil, as early, it may be, as the sixth day. The mucous membrane is either dry and shining, or coated with thick mucus, which clogs the fauces. Ulceration of any part of the throat, other than the tonsils, before the fifth day, is very uncommon in cases which are not, for other reasons, classed under the gravest forms of the disease. There can be no question that the majority of "ulcers" and "sloughs" observed during the first four or five days of Scarlet Fever are nothing but excess of the secretion of the part smeared over the surface; and likewise no question that the dire sloughing and gangrene described so well by the older writers (*e. g.* Huxham, Heberden), and on their authority still attributed at times to Scarlet Fever, appertain altogether to diphtheria. To quote the words of Armstrong: "The first four or five days there will be seldom either sloughs or specks about the tonsils; but merely an increased secretion of mucus, some of which often adheres to the part and looks like an ulcer. But when the fever continues longer, or runs higher, specks generally form about the tonsils, which are finally converted into superficial ash-coloured sloughs." Ulcerative angina, then, either occurs in cases conspicuous far more by the symptoms called malignant, or is a sequela, an epiphenomenon, supervening after the cessation of the specific disease.

The lymphatic glands at the angles of the lower jaw become enlarged and tender; in fact, inflamed. In severe cases the connective tissue around is involved, and puffy; but neither brawniness nor suppuration occurs before the rash has begun to fade.

Difficult deglutition, snoring, and alteration of the quality of the voice (it is thick and nasal), are results of the swelling of the throat. Kennedy once saw a child carried off by œdema glottidis within thirty hours from the onset of the disease.

In favourable cases the angina will recede with the eruption, and have disappeared by the seventh day, the tonsils perhaps being left rather swollen, but pale. The lymphatic glands often remain large for some time.

3. *The Pyrexia.*—The height attained by the fever has been subject to singular exaggeration; the fiery hue of the eruption, and the great dryness of skin often present, conspiring to mislead the observer. The mercury of a thermometer placed in the axilla never rises above 106° Fahr., rarely above 105°. The fever certainly runs higher than in measles, and very much higher than in diphtheria, but on the other hand does not reach the degrees which are often observed in ague, typhoid fever, rheumatic fever, and pyæmia. On the day when the eruption begins to fade the fever frequently submits to a complete crisis, as indicated by the temperature not rising above the normal for twenty-four hours; should this crisis not occur, the pyrexia is prolonged for an indefinite period. (The temperature of a healthy child may reach, but does not rise above, 99°.) In two cases the thermometric crisis occurred on the fourth day, in four on the fifth, and in three on the seventh. So, out of the cases observed by Dr. Ringer, the temperature of two became normal on the fourth day, of seven on the fifth day, and of one on the sixth day. In the rest the pyrexia was prolonged beyond the eruptive period. "The pyrexia slightly remits in the morning, and, rising in the course of the day, reaches its highest point about bedtime." (Armstrong.) A similar forenoon remission and afternoon exacerbation takes place in health. Yet the Scarlatinal Fever is continual; the remission is not at any time complete until (in uncomplicated cases) the final crisis. The severer the fever the more trifling the remission.

4. *The Alimentary Canal.*—(1) Tongue. The fur present during the invasion period becomes thicker on the second and third days, and then begins to clear off from before backwards, so as to leave the tongue deep-red, broad, smooth-looking, and dotted over with elevated papillæ; a condition sometimes met with in other diseases (especially those attended with suppuration), when a thick fur clears off quickly. Both the earlier furred and later papillated condition are often absent. (2) Vomiting during the eruptive period is rare, except in the severer forms of the disease. Constipation, easily overcome, is the rule, but diarrhœa a not infrequent exception. Tympanites is sometimes present. Thirst and anorexia occur in well-marked cases.

5. *The Respiratory Organs.*—(1) Coryza, thin or mucopurulent, may supervene at any period of this stage, and is not necessarily a grave symptom. (2) It is rare to detect the physical signs of bronchitis or pneumonia before the rash begins to fade. Cough due to the condition of the fauces is common. The frequency of the respirations is proportionate to the pyrexia.

6. *The Pulse* maintains the frequency of the invasion period, and may reach 144 in an adult, 160 in a child, or even more, without any serious import. The frequency does not increase as the disease progresses, and it falls with the temperature.

7. *The Urine.*—(1) The water is diminished in quantity. (2) Urea. In an individual case of Scarlet Fever the most trustworthy standard of the amount of urea excreted by the person in health is derived from estimations made while he is kept in bed, and upon unaltered diet, for a week or ten days after the crisis. We must exclude all observations made upon cases in which the crisis of the pyrexia did not occur within the first week; and hence, in order to obtain sufficient data, the observations should be begun upon the urine of the third day at the latest. The following cases fulfilled these conditions:—

Sex.	Age.	Weight. Kilograms.	Days inclusive.		Water. cub. cents.	Urea. grammes.	Chl. Sod. grammes.
Boy	7 yrs.	22¼	1st to 4th	pyrexia	377	14·28	2·21
			5th to 12th	apyrexia	450	15·02	4·59
Boy	5 yrs.	16½	2d to 4th	pyrexia	551	18·83	0·97
			5th to 9th	apyrexia	709	13·33	4·69
Girl	10½ yrs.	27½	2d to 5th	pyrexia	316	16·17	0·67
			6th to 13th (minus 1 day)	apyrexia	567	19·64	4·38
					per 24 hours.		

These figures suffice to show that there is no necessary increase in the quantity of urea excreted during the pyrexia. (3) Chloride of sodium is more or less diminished, sometimes very much, sometimes very little. This diminution cannot be connected with any known concurrent condition. The salt is not subsequently passed in abnormal quantity. The diminution mostly ceases suddenly on the fourth, fifth, or sixth day. (4) Phosphoric acid undergoes very decided changes in quantity, which, normal, or a little more than normal, during the first three or four days of the disease, on the fourth or fifth day is notably diminished, for the four subsequent days remains at a half or a third of the normal, and then assumes the standard of health.[1] These changes bear no relation to the temperature or any other condition yet discovered; the same phenomenon of diminished excretion of phosphoric acid at the climax of the pyrexia, or soon after, appears in ague, measles, and probably in other febrile diseases. (5) Uric acid. The only suitable case in which I was able to make a daily estimation by weight of the uric acid, manifested a great diminution (almost a suppression) in the quantity of the acid on the second and third days, a return to the normal on the fourth, and a great excess on the fifth day (the last day of pyrexia); after this the normal was resumed. It is highly probable that similar changes always occur in Scarlet Fever of any severity, as is judged from less exact observations upon several other cases. Sediments of uric acid and of urate of soda are common. (6) Pigment is not necessarily increased in quantity. (7) The occurrence of albumen during this period is considered under the head of Renal Dropsy.

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[1] These remarks are based upon daily observations by Neubauer's process in eight cases of Scarlet Fever.

8. *The Skin.*—Dryness is a frequent but by no means a constant condition: the patient may sweat; but for a sweat to follow a trivial exertion, say the movements of delirium, is a bad sign. Dryness signifies, not that the secretion of the skin is diminished, but that it is rapidly evaporated.

9. Yellowness of the conjunctivæ, tenderness over the liver, and evidences of bile in the urine, are occasionally present; and when present, devoid of prognostic value. It is rare that the spleen can be felt enlarged.

10. *Nervous System.*—An agitated manner, moderate frontal headache, and delirium, are common enough in ordinary Scarlet Fever. The delirium ("noctibus aliena loqui") which occurs during the evening exacerbation is not a grave prognostic, yet delirium is a prominent symptom of some of the worst cases of the disease.

The *duration* of Scarlet Fever is said by Valleix to be from ten to forty days. But what is the necessary duration of the specific disease, the Scarlet Fever, as distinguished from the possible duration of the illness, common sequelæ included? If we bear in mind that, in regular cases, the pyrexia often wholly ceases while the eruption still stands out well, I think we may agree with Heberden that the disease rapidly recedes after the seventh day, nay, I would add, that the specific disease even ceases then. The sore throat may abate with the eruption, or be prolonged beyond, just as the catarrh of measles may increase on the eighth day of that disease.

III. *Desquamation Period.*—After the rash has receded, certain deviations from health are observed in all but the mildest cases.

1. If the pyrexia cease by the seventh day, the pulse falls in frequency, often to below the normal; irregularity is common when the nervous symptoms of the prior periods have been well marked. The temperature often remains for a week or ten days below the point which it reaches subsequently. The urine is dilute, deficient in phosphoric acid, and often deficient in acidity; earthy phosphates precipitate, and vibriones form. All these conditions of pulse, animal heat, and urine, indicate an exhaustion of the vital energy. The tongue, if it have been characteristic (as is said), becomes, in the second week, more natural, and often assumes a delicately furred, silvery appearance.

2. *Desquamation.*—With the disappearance of the rash, the skin does not regain the characters of health; the epidermis is dry and shining, has lost its pliability, and is easily thrown into fine wrinkles. Desquamation begins in a day or two: the chin, sides of the neck, and upper part of the chest are mostly the parts first to desquamate. Where the cuticle is delicate, it becomes everywhere scurfy, and is so thrown off: where thicker it is raised and removed in small isolated patches, which increase in size by a continuous desquamation of their margin, until they meet, and so leave the whole surface desquamated; and lastly, where the epidermis is very thick, it is undermined in large flakes before it is cast off. The amount of the desquamation

depends very much upon the previous intensity of the rash: where the latter has been slight, there the former may be almost absent. Miliaria cause an abundant and early desquamation. The duration of desquamation is from a few days to a month or two.

3. The throat, in favourable cases, loses its redness; but the tonsils may remain swelled for some time: in like manner the lymphatic glands cease to be tender, and gradually diminish in size.

Such is normal or regular Scarlet Fever; it remains to trace the disease to its extremes, of severity on the one hand, and of mildness on the other. And first of Scarlet Fever of abnormal severity, commonly called

MALIGNANT SCARLET FEVER.

But before going further I must enter my protest against the manner in which the epithet "malignant" is used, or rather abused, with regard especially to Scarlet Fever, but also more or less to many other diseases. Malignity has almost ceased to have any real meaning, and all that some of the greatest physicians have written upon the subject seems to have been written in vain. This is not the place for me to discuss what malignity really intends, however much I may feel inclined to do so: in what follows I shall simply strive not to misapply the term in the case of Scarlet Fever.

The severer forms of Ordinary Scarlet Fever merge into a type of malignity characterised by excitement, followed by exhaustion. There are all grades of severity between this, the least grave form of malignity, and that which places Scarlet Fever almost on a footing with Asiatic cholera and the plague, that form in which a preliminary period of excitement is hardly to be perceived, so rapidly does collapse follow upon the onset of the disease.

Type I.—The malignity is deuteropathic, secondary to a state of ataxia or excessive disorder of the functions of the nervous system; in the nomenclature of Armstrong the malignity is inflammatory. The invasion symptoms are all well marked, yet even from the first, those which are to be referred to a disturbance of the nervous system predominate,—delirium, agitation, sleeplessness. The premonitory stage over, the rash comes out well, the sore throat is considerable, the fever runs high. Delirium remains, exists at all times, and is active; the patient throws himself about, gets out of bed, sings loudly; his mind is confused; his eyes bloodshot; pulse frequent, full and soft; extremities warm; vomiting after food is a prominent symptom; there may be diarrhœa or coryza. But depression follows in a day or two, the delirium is much less active, the person lies muttering, the pulse becomes small, weak, and still more frequent, the face gets dusky, the cutaneous circulation is remarkably sluggish, swallowing is imperfectly performed, vomiting may persist, the belly is tympanitic. Lastly, stupor supersedes delirium, and death ensues. Sometimes the fatal issue is accelerated by a sudden attack of convulsions, the pulse ceases to be perceptible, the whole surface is blue, coma follows.

Type II.—The malignity is protopathic (congestive in the phrase of Armstrong), the vital debility is primary and spontaneous, the disease goes straight to the source of life. The patient becomes suddenly pale and faint, vomits; the vomiting is repeated with short intervals, the bowels are relaxed, the hands twitch, there are great anxiety and feeble delirium. In a very few hours the depression is extreme, the whole exposed surface feels cold, yet the temperature of the armpit is febrile; the pulse is very small and frequent. Perhaps, for a time, the patient seems to rally; he sits up in bed, but his pulse remains very frequent, and his feet cold: a faint rash may appear. Depression returns, the face becomes pale livid, and perfect stupor (interrupted by convulsions) supervenes; pulse is excessively weak and frequent, respiration remarkably irregular, sweats break out, the skin is cold and mottled; these symptoms precede death. The duration of the whole illness may be less than twenty-four or even less than twelve hours.

The temperature of the body in the rectum and axilla is maintained at a tolerably high fever standard, but I have not yet observed that extreme elevation of temperature which is to be found in some cases of rheumatic fever, tubercular meningitis, sun-stroke, and injury to the brain. It is not given to any physician to observe with sufficient closeness many cases of protopathic malignity in Scarlet Fever; by far the most uncommon form of the disease. A child who died in this manner twenty hours from the onset of illness, afforded a temperature in the axilla of only 104° an hour and a quarter before death. Half an hour previously the temperature had been 105°: this was the highest degree observed though the temperature was taken at intervals of about two hours. It is manifestly untrue to say that the vital debility is due to hyperpyrexia; they would be merely concomitant conditions in any case in which they should both occur.

Excluding the examples of these well-marked types of truly malignant Scarlet Fever from the whole medley of cases which are loosely called malignant, there is left a large and heterogeneous caput mortuum which consists in part of cases attended by unusual or unusually severe local lesions, and in part of cases which certainly may lay a sort of claim to be regarded as instances of a prolonged malignity. The latter class of cases runs a course more or less of the following kind. For the first day or two the attack does not seem to be unusually grave; but about the third day the patient is alternately restless and heavy, pulse frequent and feeble, expression vacant, face pale and thick-looking, mouth very tender, sweats follow a slight exertion; coryza, diarrhœa, tympanites, ulceration of the tonsils, and dysphagia, supervene, some or all; delirium is frequent, emaciation proceeds rapidly, and the patient dies exhausted, it may be during the first week, or not till far on in the third; the prolongation being due, partly to the local processes set up, and partly to inability to rally.

The rash in Malignant Scarlet Fever is of small prognostic value; it is often abundant and of a bright colour; in the worst cases no rash appears, but none would be expected, death occurring before the second day. The tonsils mostly ulcerate early, even on the first day. The pyrexia lasts to the very end.

LATENT SCARLET FEVER.

The symptoms are so ill-developed as to be not characteristic or not observed. Examples of Sydenham's Scarlet Fever—disease by name alone—hold a mid place between this latent and the regular forms. How mild soever the primary disease, the gravest sequelæ may ensue.

SEQUELÆ.

As symptoms of the desquamation period, I have described sundry sequelæ of Ordinary Scarlet Fever which are constant, and the necessary results of normal precedent conditions: there remains for consideration a long list of sequelæ which are inconstant in their occurrence, and therefore mostly looked upon as complications of the disease. The distinction may be somewhat artificial, but it is in the present state of knowledge convenient.

I. *Sore-throat.*—Any sore-throat which may be present during the decline of the rash or after it, is due either to a simple persistence of the ulcerated condition of the tonsil, which has been described as an occasional symptom of the eruptive period, or to the establishment of a new form of lesion. In the former kind of sore-throat, the ulceration tends to progress deeply rather than widely, and bubo is a frequent concomitant. The latter kind supervenes most frequently on the sixth day, sometimes a day or two later, but never after the end of the second week: in very many cases the fauces have previously escaped grave implications; then, at the time specified, one or several small greyish patches, surrounded by a vivid red margin, appear on the tonsils, uvula, or arches of the soft palate, sometimes on the hard palate or the tip of the epiglottis. At the same time the fauces are swelled and reddened, deglutition gives pain, the lymphatic glands at the angles of the jaw become larger and tender, the connective tissue is puffy. The epithelium of the tongue is sometimes raised in patches. The excoriations go on to heal or to ulcerate. If to heal, the pellicle becomes thicker, so as to look like a false membrane, the surrounding redness abates, and in a short time, when the scab is thrown off, the mucous membrane is left uniform in surface. In the other alternative, when the pellicle is removed, the deeper layer of the mucous membrane is left exposed, ulceration ensues at this spot, and thence may spread far and wide; in the worst cases the tonsils are destroyed, the cartilage and bone in the vicinity are laid bare, dysphagia ensues, the fluids return through the nose. The state of the sides of the neck corresponds: in the favourable cases the swelling gradually disappears; in the severe, all distinction between gland and connective tissue becomes lost, a "collar of brawn" extends around the neck (scarlatinal bubo), softening ensues at places, and here the skin will slough in order to give exit to a shreddy sero-pus; yet the swelling is not diminished until large sloughs of the connective tissue come away, and the skin (it may be of the whole neck) is left floating over the parts beneath, and perforated in numerous holes. The suppuration reaches downwards into the mediastinum, or spreading upwards, invades the parotid glands, passing along the interstitial connective membrane, and dissecting out the salivary lobules. Hæmorrhage from the large vessels of the neck sometimes occurs to increase the complication. The rapidity with which the hard brawny swelling will establish itself is remarkable: a neck which was only puffy one day may be hard the next; a semi-comatose condition is often produced: the veins of the eyelids are turgid. The majority of cases of extensive sloughing of the connective tissue die; symptoms not directly connected with the throat are apt to supervene, particularly vomiting and diarrhœa. Simple suppuration is a much less grave result; and indeed a very considerable amount of firm swelling of the neck may be resolved without suppuration.

II. *Coryza.*—Coryza, which persists into or supervenes during the second week, is an important symptom, indicative of a morbid state of the mucous membranes in general. The direct consequences of coryza are often serious, and especially the extension of disease along the Eustachian trumpet into the tympanum; when this cavity is filled with pus, the membrane bursts or sloughs, and an obstinate otorrhœa results. Fistula lachrymalis is much less common. Worst of all is the coryza, which is indicative of nasal diphtheria, a complication comparatively infrequent.

III. *Otorrhœa.*—Otorrhœa is of two kinds. The first is that otorrhœa which begins in the external meatus; the discharge, which is whitish at the onset, soon becomes purulent, while the lining membrane everywhere thickens. The second kind of otorrhœa is that which follows coryza; ulceration of the lining membrane of the tympanum, necrosis of the bone, facial paralysis, incurable deafness, and abscess of the brain, these are consequences not uncommon: the discharge is purulent from the first; when injections are used, they pass from the nose out at the ears, and the reverse. Hæmorrhage from the internal carotid artery is a rare sequel.

IV. *Diarrhœa.*—This sometimes coincides with the decline of the rash. Bloody stools and excoriations of the anus and buttocks may be the consequence of this, as of other kinds of diarrhœa.

V. *Bronchitis and Pneumonia.*—Complications more common in the second than the first week, and mostly preceded by coryza. Severe bronchitis is not necessarily fatal; when it is fatal, we always find lobular pneumonia superadded. Primary lobar pneumonia sometimes occurs.

VI. *Abscesses.*—Convalescence is often put back by the formation of one or more abscesses; if they are very large or numerous, they may be the immediate cause of death, and even when they are small and solitary the local results are sometimes grave. For example, on the back of the hand, an abscess as it closes leaves the tendons glued to the neighbouring parts. Or again, behind the pharynx, an abscess may cause death, especially in infants. The symptoms of post-pharyngeal abscess are dysphagia, return of fluids through the nose, some stiffness of the neck, one side of which may be fuller than natural, and dyspnœa. Any one, or even all of these symptoms, may be so slight as not to attract attention. The finger passed into the pharynx feels a soft tumour projecting in the middle line or somewhat to one side. Should the abscess burst spontaneously, sudden suffocation may ensue. Abscesses about the neck are nearly always, if not always, the result of internal ulceration.

VII. *Rheumatism.*—Towards the end of the second or beginning of the third week, we occasionally observe a considerable increase or a re-establishment of the pyrexia, and at the same time an affection of the joints; the latter consisting in great tenderness and elastic swelling (more around than in the joint), with or without redness of the skin; the patient sweats at times, perhaps profusely; the serous membranes are prone to inflammation. There are all grades of severity between transitory pain in a single joint and painful swelling of nearly all the joints of the body. A similar complication may attend the eruptive period, but is less common; it is common enough for adults to complain of great tenderness of the muscles while the rash is present. The swellings are mostly resolved, and then the patient recovers; but sometimes suppuration occurs around and in the joint. Suppurative rheumatism is fatal. The nature of this complication must, at present, be left an open question, whether the disease be really rheumatic, or whether it be pyæmial.

VIII. *Renal Dropsy.*—Scarlet Fever is very often followed by albuminuria, dropsy, and a form of nephritis; three accidents which it is convenient to group together and to view as collateral symptoms of a more general condition.

1. *Condition of the Urine.*—The proportion of cases of albuminuria seems to differ with different epidemics: albumen was at no time present in the urine of six out of twelve children, the whole of whose urine was saved and examined daily by myself, from the decline of the rash to the fourth week; Abeille found albuminuria in one-third of the cases examined by him; in the experience of Begbie, Newbigging, and Holder, the occurrence of albuminuria was a rule without an exception. In twelve cases I tested the urine daily throughout the fever and the convalescence; in one, albumen appeared on the fourth day, in one on the eighth, and in none was albumen detected for the first time after the twenty-first day; four cases occurred during the second week, seven during the third. In eight out of twenty-one cases observed by Abeille, the urine became albuminous before the sixth day; Jaccoud has noticed albuminuria as early as the second day. In the two above-mentioned instances of the occurrence of albuminuria

on the fourth and eighth days, the abnormal condition of the urine was present for one day only; in the ten other cases the duration of the albuminuria was at least a week. In most of the examples of the more enduring form of the disease it is easy to distinguish three stages, each characterised by a peculiar condition of the urine.

Stage 1.—The quantity is diminished (total suppression for several days has been observed by others now and then), the specific gravity is increased in proportion to the concentration, urate of soda is precipitated, the colour is unchanged, and, when any saline deposit is dissolved, however turbid the urine be with organic matters, it is not at all smoky: the urea is diminished in quantity beyond the proportion of the diminution of the water; the relative lack of chloride of sodium is greater still; albumen is present; when the diminution in the quantity of urine is considerable, the microscope discovers casts, some clear and perfectly free from granules or epithelium, some finely ganular, but hardly any blood disks or renal epithelium; when the disease is less severe (and in all cases as this stage is passing away), there are renal epithelium and epitheliated casts, with a few blood disks. The amount and the duration of the diminution of the quantity of the urine in this stage are to a great extent prognostic of the future course of the disease.

Stage 2.—The quantity of urine regains the normal, and soon exceeds it; there is a perfect diuresis; the specific gravity falls low; the urea and chlorides return to the standard, or nearly so; the urine becomes bloody (from slight smokiness up to the deepest brown); the amount of albumen is relatively less, though it may be absolutely greater than in the earlier stage, and is not proportionate to the depth of the colour of the urine; the microscope shows blood disks, hæmatoidin crystals (?), renal epithelium, epitheliated and granular casts. As this stage passes off the urine becomes clearer, less red, and more yellow (passing through brown and green); the albumen lessens.

Stage 3.—The diuresis continues, but the smokiness disappears; though dull with excess of organic flocculence, the urine is normal in colour; the albumen gradually disappears. Even after albumen has ceased to be present, it mostly happens that for some time the quantity of urine secreted is above the normal.

In mild cases the distinctions of the stages are necessarily ill-marked; sometimes the urine is at no time diminished in quantity; sometimes, although the first stage is well marked, the urine is hardly at any time smoky; sometimes the third stage never comes to an end, or in other words the urine remains permanently albuminous; a sudden increase in the depth of the bloody colour of the second stage often occurs, without the quantity of albumen in the urine being proportionally increased; the urine returns to its prior condition in two or three days. A class of cases, by far the worst of any in a prognostic point of view, has been exemplified by several children who, when first they came under observation, brought a history to the effect that two or three months previously they had had Scarlet Fever (perhaps so mildly that the nature of the illness was ignored at the time), and that after the lapse of several weeks dropsy had supervened, not suddenly, but gradually, increasing from day to day. The urine is normal in colour and specific gravity, clear or with some excess of flocculence, not necessarily diminished in quantity; but the amount of albumen present is very large; very little is to be seen by the microscope, and that little is not characteristic. The steps by which this stage is reached have not yet been observed; so far as the writer has seen, the condition is permanent and resists all treatment.[1]

2. *The Dropsy.*—Dropsy or hæmaturia is the first symptom which arrests the attention of the inexpert. The former is very uncertain in occurrence, and is often almost wholly absent from first to last; when it is the first symptom noticed, it supervenes rather suddenly in the third week, and especially towards the end of that week. Dropsy is mostly preceded by albuminuria for a day or two; occasionally the dropsy precedes the albuminuria; very rarely, when both symptoms have been present, the albuminuria nearly wholly (possibly wholly) ceases, and yet the dropsy remains. In some epidemics, dropsy without albuminuria at any period is common. Philippe of Berlin did not once find the urine albuminous in more than sixty cases of scarlatinal anasarca (quoted by Jaccoud). The dropsy assumes the form of—(1) Anasarca, which may be general or limited to the eyelids, backs of hands, and bottom of back: œdema glottidis is a very rare result of scarlatinal dropsy. (2) Serous dropsies, which occasionally precede the anasarca: when peritoneal, the effusion is of no great practical moment: the diagnosis of pericardial effusion depends upon physical examination; dyspnœa, intermitting lividity, indistinctness of the pulse, and a tendency to syncope, may or may not be present: pleural dropsy, hydrothorax, is a more common and formidable complication. A child whose anæmia and anasarca have shown a tendency to increase, and who has a slight cough and some sonorous rhonchi over the lungs, suddenly becomes much worse, vomits repeatedly, dyspnœa and lividity ensue and rapidly increase, very little urine is passed; the distress is painful to behold; and death rapidly follows, mostly within twenty-four, sometimes within six hours from the first exacerbation of symptoms. *Post mortem* both the pleural cavities are found to contain a great excess of serum, which is perfectly clear, or floats the network of a delicate coagulum; sometimes lymph is present, the evidence of pleurisy hereafter to be described. The lungs are collapsed but œdematous also, dark iron-grey on section, and capable of imperfect insufflation.

3. *General Symptoms.*—(1) Pyrexia accompanies the onset of renal dropsy, and is high in proportion to the severity of the symptoms: in mild cases pyrexia is almost absent: it is rarely prolonged into the second stage of albuminuria. All the more chronic cases of renal

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[1] Soon after the first edition of this volume was published, Dr. Weber read a paper upon some cases of this kind before the Medical and Chirurgical Society. Transactions, vol. xlix. p. 199.

dropsy are, when uncomplicated, apyrectic. The attendant symptoms are heaviness, dry skin, troublesome vomiting, parched lips, and constipated bowels. (2) Anæmia is mostly a marked symptom after the pyrexia has passed off. Children who are dropsical look more pale than those who are not. (3) Uræmia: convulsions and coma, supervening upon scarlatinal dropsy, are more often symptomatic of the onset of some local inflammation, than indicative of uræmia. Indeed true uræmic convulsions are not very common; when they do occur they induce a fatal issue in a minority of cases: on the other hand the prognosis is most unfavourable when the convulsions merely assume the place of a rigor.

The cause of renal dropsy is unknown: that exposure to changes of temperature will insure dropsy when the urine is albuminous (or rather, perhaps, when that condition which tends to produce albuminuria is present), has been admitted by most observers; but that a chill is a frequent cause of albuminuria is a dogma much more disputable, and apparently disproved by the fact that albuminuria is a sequela almost constantly present in some epidemics and almost constantly absent in others.

IX. *Serous Inflammations.*—They mostly, not always, are accompaniments either of albuminuria or of the rheumatic state. 1. In the latter, the pericardium is especially prone to inflame, a complication which, by itself, does not render a prognosis unfavourable. 2. In albuminuria, pleurisy is the most common. One or both pleuræ are inflamed, the lymph effused rapidly breaks up into pus, and, in most cases, death ensues in a few days; yet occasionally a chronic empyema is the result. States intermediate between pure hydrothorax and pleurisy are sometimes met with. Pneumonia is frequently combined with the pleurisy. In the pericarditis and peritonitis likewise the lymph has a great tendency to become puriform. It is not rare to find all the serous membranes inflamed at once. 3. Occasionally pleuro-pneumonia, pericarditis, or peritonitis supervenes, and there are neither dropsical nor rheumatic symptoms present. Yet such sequelæ are especially apt to occur in what may be termed the rheumatic period; that is, the second week. 4. Systolic cardiac murmurs sometimes spring up during the illness: they are heard with greatest intensity at the apex or the second left interspace. Neither albuminuria nor rheumatic symptoms necessarily accompany the development of the apex murmur: nay more, it can hardly be said to be proved that the occurrence of a systolic murmur at the apex of the heart is always due, in these cases, to endocarditis. These apex murmurs in my own experience persist for a month or two at least,—persist as long as the patient is under observation. Sometimes, possibly, dilatation of the heart originates in an attack of Scarlet Fever.

X. *Other Sequelæ and Complications.*—(1) Sloughing of the cornea is an accident of which the occurrence may probably be always prevented. (2) Hæmorrhage in consequence of a sloughing bubo, of destruction of the pars petrosa, or in the form of hæmaturia, has already been described. Epistaxis is an occasional phenomenon. Here mya be mentioned that variety of Scarlet Fever which many writers, probably somewhat biassed by the analogy of small-pox and measles, have been very ready to admit into their nosology, I mean Sc. hæmorrhagica; a form of the disease which must be very rare (except, perhaps, in certain epidemics),—so rare, that the occurrence of a passive hæmorrhage from several mucous membranes at once might make one with justice suspend a diagnosis of Scarlet Fever until the notion of possibly having to do with variolous roseola was discarded. I have known Variola hæmorrhagica to be mistaken for Sc. hæmorrhagica, and the truth not appear until, in course of time, those who had dealings with the case were themselves attacked by small-pox. (3) Gangrene—Cancrum oris is a very uncommon sequela of Scarlet Fever. Gangrene of the pharynx is said to have occasionally supervened. Sloughing of the skin over a bubo is much more common; so also is gangrene of blistered or ulcerated parts of the skin. Here may be classed such sequelæ as necrosis of parts of the jaw-bones, and hip-disease. (4) Tubercle. Scarlet Fever by no means tends specially to develop tubercle even in a subject predisposed. (5) Other acute specific diseases. The eruptions of Scarlet Fever and measles may appear at the same time upon the same patient (Rilliet et Barthez, iii. 281). Small-pox has been known by the same writers to complicate Scarlet Fever. I myself have made this observation: a girl of three had hooping-cough; about a week after she began to whoop she became very feverish,—this was on April 5th; on the 6th and 7th she vomited beyond what the cough would account for; on the 8th an indubitable varioloid eruption appeared upon her, and on the 9th, a rash which possessed in every respect the scarlatinal character. In another girl of two and a half I saw the eruptions of Scarlet Fever and varicella appear upon the same day. I have known the long course of typhoid fever to be broken asunder, as it were, by an attack of Scarlet Fever. Diphtheria is a comparatively frequent sequela: the patient seems to be in a fair way of recovery, when an acrid discharge from the nostrils is noticed, the neck swells again, the pyrexia returns: death is inevitable (Graves, Clin. Med. i. 318; Trousseau, i. 15): this complication may ensue as early as the fifth day (Rilliet et Barthez, iii. 168). The writer has seen diphtheria supervene upon the chronic dropsical cachexia, the new disease here also inducing rapid death. Lastly, Scarlet Fever may be intercurrent during the acute period of rheumatic fever, and neither disease be perceptibly modified.

DIAGNOSIS.—During the invasion period the diagnosis depends upon the presence of vomiting or angina, and the absence of sneezing, lacrymation, or pain in the back, in a person who has been taken ill suddenly, and has not previously suffered from Scarlet Fever. The frequency of the pulse, and severity of the nervous symptoms from the first, are sometimes valuable aids to diagnosis.

The rash may be confounded with the eruption of measles, small-pox, typhus, roseola, miliaria, or urticaria. Measles may be wrongly suspected when a Scarlet Fever rash is of a darker colour than usual, and especially when it is discrete also. And, contrariwise, in several cases of measles I have been perplexed by the existence of a scarlatiniform rash upon the body several hours before the proper eruption of measles appeared. The roseola which precedes small-pox occasionally causes an error in diagnosis. The resemblance which the rash of Scarlet Fever occasionally bears to that of typhus has been alluded to previously. To know that the minute vesicles of miliaria and the confluent wheals of urticaria are not unfrequently mistaken at first sight for Scarlet Fever rash, will suffice in most cases for the diagnosis. Reviewing some of these statements, and bearing in mind how closely the rash of Scarlet Fever and of measles may simulate each other, that the two diseases may concur, and that symptomatic roseola is not always easily distinguished from Scarlet Fever on the one hand or measles on the other, it is fair to conclude that at least some supposed cases of a disease which has been called rubeola (rötheln, roseola febrilis) are to be attributed to an impossibility in the diagnosis.

Scarlatinal angina is indistinguishable *per se* from ordinary simple erythematous sore-throat, or from the pellicular angina (pharyngeal herpes) which forms so large a proportion of the sore-throats popularly styled diphtheritic.

The swelling which occurs about the joints during the first two or three days of the disease is sometimes very considerable, while the rash is pale or absent: such Scarlet Fever has been mistaken for rheumatic fever.

Surgical Scarlet Fever.—It has been doubted by some whether the scarlatiniform rash which sometimes follows operations is really scarlatinal. The eruption appears from the second to the sixth day after the operation, and, in the cases which have caused the doubt, is very fugitive, the first and only symptom. Yet, that the disease really is Scarlet Fever would seem to be proved by the following observations: first, that the disease occurs in epidemics; secondly, that in a given epidemic a severe case occasionally relieves the monotonous recurrence of the very mild form; thirdly, that a precisely similar scarlatinilla attacks, in the same epidemic, patients who have not been subjected to operation, and who have no open sore; and lastly, by way of a veritable *experimentum crucis*, that, however freely these patients are exposed to Ordinary Scarlet Fever contagion afterwards, they do not contract that disease.

MORBID ANATOMY.—Scarlet Fever does not possess any distinctive anatomical character which persists after death: the alterations of texture found *post mortem* are common to other pyrexiæ.

The furred tongue indicates a condition, similar in kind if not in degree, of the mucous membrane of the whole alimentary canal; there is an excessive formation of epithelium and concomitant hyperæmia of the sub-epithelial layers; casts of the gastric tubuli are to be detected by the microscope in the vomit. (Fenwick.) From the frequency of coryza, pulmonary catarrh, and pneumonia, we infer that the respiratory mucous membrane does not escape.

A peculiar change in the muscular tissue has been described by Zenker; a change more constant, as it would seem, in typhoid than in Scarlet Fever.

The puffy swelling which sometimes precedes the rash or coincides with it, may indicate a direct implication of the connective tissue.

The spleen, the lymphatic glands, the tonsils, and the lymphoid (solitary or agminated) glands of the stomach and intestines are swollen and injected; the tonsils and the gastro-intestinal glands sometimes go on to ulcerate. In this place may be just mentioned the leuchæmic deposits which have been detected here and there.

The liver and the kidneys are involved to a slight degree; a little cloudiness of the cortex of the kidneys by no means indicates the existence of albuminuria. The kidneys of renal dropsy will be described under the head of Kidney Diseases.

The cerebral symptoms are, so far as is known at present, unaccompanied by anatomical change.

PROGNOSIS.—I. *Prognostics derived from pre-existing conditions.*—1. The social position of the person attacked has no influence upon the course of the disease, which is quite as fatal among the rich as among the poor. 2. Family constitution seems to influence Scarlet Fever to a degree which can hardly be overrated; that one or more members of a given family have succumbed, renders the prognosis in the case of any other persons in that family who may chance to be attacked, very grave. 3. Pregnant women are said to be peculiarly exempt from the liability to contract Scarlet Fever; but the puerperal state predisposes to the occurrence of not only Scarlet Fever, but Scarlet Fever of a very fatal form. The rate of mortality seems to differ in different epidemics; but whether of thirty-six patients attacked all die (Trousseau), or the mortality be twenty-five per cent. (MacClintock), or all of nine patients recover (Blakely Brown), the prognosis cannot be too guarded. 4. A scrofulous vulnerability of the mucous membranes or the lymphatic glands greatly adds to the gravity of the prognosis. 5. A previously feeble state of health by no means predisposes to a bad type of Scarlet Fever—almost the reverse; certainly the majority of cases of Sc. maligna are in persons of previously robust health. 6. Age and sex have no effect. The epidemic constitution does not always afford trustworthy aid in a particular case.

II. *Prognostics derived from the actual disease.*—The number and the gravity of the possible complications and sequelæ of Scarlet Fever should render the prognosis very wary even in the mildest cases. Excluding the malignant forms, we judge of the severity of the disease more from the number and severity of the local lesions, than from the height of the pyrexia or the colour of the rash; each additional local lesion, complication, or sequela, being an addition to the gravity of the prognosis. Bubo and coryza are the most formidable symptoms of the first ten days, rheumatism and albuminuria of the next ten days. There are no guides to prognosticating the probability of the occurrence of bad sore-throat, rheumatism, or renal dropsy. If on the first day of deflorescence of the rash the fever still keeps up, we must attribute it to a new or an increased local lesion, and this will mostly be found to be anginal. The gravity of a brawny bubo is always great. A tendency to relaxed sore-throat does not predispose to bad angina. In most cases in which coryza has appeared on the fourth or fifth day, the secondary sore-throat will ensue to a certain extent. Of the patients seen by MM. Rilliet and Barthez, all who, during the first fifteen days of the disease, exhibited convulsions, convulsive movements, rigidities, in other words, symptoms referable to the locomotive apparatus, have died; according to my own experience, the prognosis which might be derived from this statement would be too unfavourable. The nocturnal talkative delirium (noctibus aliena loqui) is, no doubt, as Heberden says, an unimportant symptom. But when delirium becomes more active than this, more constant, more independent of surrounding things; when attended by restlessness, sleeplessness, drowsiness, or by vomiting and diarrhœa, it is impossible to dissemble the grave significance of the symptom. Suppurative arthritis is a most serious accident; multiple abscesses are much less grave. In the renal dropsy, moderate œdema pulmonum and pleural effusion may be recovered from. The reader will find many other prognostics under the heads of the sundry symptoms.

PROPHYLAXIS.—Removal from all sources of contagion is the most obvious, the surest, and probably the only means of preventing Scarlet Fever. The prophylactic virtue of an infinite number of fumigations and drugs has been vaunted from time to time, but fruitlessly, with one exception, namely, belladonna; yet even of that remedy the reputation, not wanting the support of hundreds of observations tabulated, has in the present day sunk very low. Inoculation by means of the blood, the fluid of the miliaria, or the secretions of the fauces, has been practised in a few cases, and so far, apparently, not without favourably modifying the disease thereby communicated.

With reference to the purification of materials which have been exposed to scarlet fever, it may be here remarked that the morbific principle is destroyed by a heat considerably below the boiling-point of water.

TREATMENT.—In the following pages I shall endeavour to trace an outline of the plan of treatment ordinarily adopted at the present day. It will be impossible not to omit mention of many curative means which may be of real value; in a common disease, not very powerfully controlled by any therapeutic method, the remedies which have been and which are employed must be innumerable.

To know the natural course of Scarlet Fever is a great guide to the appropriate treatment; for, although we cannot abridge that course, we can be prepared for the occurrence of different accidents at different stages, and so be able to meet those accidents from the very first. Provided that no complications occur, Scarlet Fever will terminate favourably within a week from the onset of the disease. Among the complications we must place the malignant form of the disease, and also any unfavourable pre-existing condition, such as the puerperal state.

The regimen of acute diseases must be put in force. The patient should be kept in bed, as a rule to which there is no exception; the bed-clothes should be those to which he has been accustomed in health, and no more; carpets, curtains, and porous materials must be removed; the bed-room should be carefully ventilated (in part by an open fire), bearing in mind that there is no special reason to fear cold during the first week; the whole surface of the body should be sponged with tepid water once or twice a day, and subsequently to grease the skin with mutton suet often brings comfort to the patient. The diet is to be unstimulating, consisting of milk, broths, the farinacea, an egg, light puddings; drink should be freely supplied. Purgation is to be avoided.

In many cases a consideration of the previous and the present condition of the patient will indicate the administration of wine: the child seems low, the pulse is not only frequent but soft and feeble, there is possibly coryza present. At the same time full doses of carbonate of ammonia should be given, in milk, every four hours. And it must be conceded that no great harm comes from the moderate employment of stimuli, even when they are not absolutely necessary.

When the throat is much inflamed, great relief may be afforded by either of the following methods of treatment. The first is to cause the patient to use ice freely; he is to allow lumps of it to dissolve in his mouth. Under this treatment tenderness of the submaxillary glands sometimes passes away in a few hours, an improvement which is an index of that which has taken place within. Ice can be easily given to patients above five or six years of age; but the second remedy is one which is inapplicable in the case of children; I mean the inhalation of the steam of hot water. Puffy swelling of the neck may often be removed by external applications of spongio-piline wrung out from hot water, or of hot linseed-meal poultices, frequently renewed.

In like manner coryza is a symptom which, when treated early, may thereby cease to be of evil omen. To check the coryza is to check, in the majority of cases, that worst form of otorrhœa which proceeds from the destruction of the tympanum. In patients above eight years of age, the valuable method of washing out the nostrils introduced by Dr. Thudichum may be employed. An ounce of salt is dissolved in a pint of warm water, and this solution, contained in a vessel a little raised above the head of the patient, is conveyed by means of a flexible caoutchouc tube into one nostril; respiration being carried on through the mouth, and all attempts at swallowing forbidden, the fluid passes out freely by the other nostril.[1] In young children we are reduced to the expedient of syringing the nasal fossæ with a weak solution of nitrate of silver (gr. v. to ℥j.) once a day, when the coryza is troublesome.

Of the remedies employed in the treatment of the malignant form of Scarlet Fever, there is one which stands out from among the rest, the cold affusion. Yet it is not of equal value in all cases; from the days of Currie downwards the ataxic form of the disease, characterised by delirium, diarrhœa, vomiting, full pulse, and great heat of skin, has been recognised as the special indication for this active treatment. The patient is to be seated naked in a bath, two or three bucketfuls of water at 70° F. are poured over him quickly, so that the affusion does not last longer than half a minute; he is then returned undried into bed, and laid between blankets. The first affusion having had a markedly beneficial effect, should the indication symptoms return in the course of the same day or the next, the water treatment may be repeated, and this even two or three times if necessary. When this treatment has been objected to, or has seemed too bold, I have seen very good results follow from packing the patient in a wet sheet for an hour. A still milder method remains to be mentioned, that of occasional cold sponging. Ammonia and brandy are nearly always needed, sooner or later, by these patients.

In the primary adynamic form all treatment will be baffled. The cold affusion is the only means which has seemed to me to be of even momentary benefit. Hot mustard baths, which would at first sight appear to fulfil the indication better, may be tried; strong tea or coffee, brandy, ether, camphor, are to be given internally. The treatment is much the same, and also, unhappily, the prognosis, in adynamia, sequential to an excited state. A full description of the treatment formerly adopted (and not, perhaps, without success) in cases such as these, will be found in the pages of Armstrong.

In the more prolonged adynamia, quinine is a useful addition to the remedies just mentioned. A most nutritious diet and a rather liberal allowance of wine will often enable such patients to recover.

The Hippocratic remedy of warm affusion to the head is most soothing in the nocturnal delirium and sleeplessness.

However favourable an attack of Scarlet Fever, the patient should be kept in bed for three weeks from the commencement of the disease; he may then get up, but he should not leave his room for another week. Even after four weeks have elapsed he is not free (if we are to believe some observers) from all danger of albuminuria.

If on the morning of the fifth or sixth day any ulcerous appearance that the fauces may have previously presented does not show signs of yielding, it is well to cauterize the morbid surface. For the tonsils undiluted hydrochloric acid is to be used; for any other part of the soft palate, solid nitrate of silver. The latter caustic is to be applied to those excoriations which are apt to appear about this time or later. It need hardly be added that these potent escharotics are as a rule to be used once for all; if repeated, four or five days at least should have elapsed since the previous application. The external swelling should be assiduously fomented while in the puffy stage, for as soon as brawniness sets in, anything that may be done will be of very small service; poultices are to be continued, but the occurrence of suppuration will hardly be thereby hastened. As soon as suppuration has occurred, however small the spot to which the process is limited, an incision should be made to let out the pus, the poultices being afterwards resumed. The abscess should never be allowed to open itself. In the worst cases of Scarlatina anginosa, openings and counter-openings will be required; a free discharge of sloughs and ichor affords the patient his sole chance. Should hæmorrhage occur, the wound is to be stuffed with lint soaked in the solution of the perchloride of iron; this moderate pressure will stop the bleeding, which is more often venous than arterial.

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[1] For fuller particulars refer to Dr. Thudichum's paper. Lancet, Nov. 26, 1864.

All cases of otorrhœa are to be treated by syringing the meatus gently with warm water three or four times a day. Should a discharge either from the ear or from the nose become chronic, quinine and sulphuric acid are the drugs indicated.

The suppurative tendency likewise is an indication for quinine, and an abundance of fresh air and substantial food. All abscesses are to be opened early. A post-pharyngeal abscess should be evacuated if possible (and it mostly is possible) through the neck; should this be impracticable, a cut must be made with a guarded bistoury through the posterior wall of the pharynx upon the vertebral column—an operation devoid of risk, if performed with ordinary care. The matting together of tendons is remediable to a great extent by fomentations, friction, and passive motion.

The treatment of the rheumatism is that of its symptoms: Dover's powder for the pains, diluents for concentrated urine, aperients if necessary, cotton wool or poultices round the affected joints. Alkalies are not of any special value. The treatment of intercurrent pericarditis is often difficult; whether local blood-letting is admissible, and when counter-irritation more or less severe should be used, depend entirely upon circumstances beyond the local inflammation.

The remedies to be employed at the onset of renal dropsy may be thus arranged in order of importance. First come active purgatives: elaterium is good, but the uncertainty of the ordinary drug often causes the loss of much valuable time; compound jalap powder in doses of not less than a scruple to a child of six or eight years of age, repeated at intervals of eight hours, until the bowels act freely, is altogether more trustworthy. The hot-air bath, preceded by a hot-water bath and a dose of antimonial wine, may be used every night so long as the quantity of the urine is much diminished. After four or five such baths the strength of the patient should be carefully estimated, and the baths continued or not accordingly. Dry-cupping, not practicable in the case of children, would no doubt deserve trial in older patients. Vomiting is an intercurrent symptom which will perplex the practitioner; not the least valuable remedy will be found to consist of ice swallowed in the lump. Diluents should be given as much as possible. It may as well be observed that there is a concentration of the urine which is due to insufficient ingestion of liquids or to excessive sweating; to drink water freely is all that is needed in such cases to increase the quantity of urine.

The less acute stage of albuminuria (called the second in the previous pages) requires an altogether different plan of treatment. The bowels must not be confined, but purgation is no longer necessary. Hot-air baths have little or no beneficial effect upon the condition of the urine. When there is much hæmaturia, gallic acid in sufficient doses may be tried; it should be discontinued if it does not bring about a decided improvement in four or five days; to diminish the quantity of albumen as such, gallic acid has little if any power. Quinine has at times a remarkably good effect in these chronic cases. But upon the whole no remedy is equal in value to the perchloride of iron. Counter-irritation to the loins will be useful, and generous diet necessary. In the great majority of cases the disease tends to wear itself out in time. When extensive anasarca is present, the whole condition is more grave; should the dropsy tend to the lungs and pleural sacs, the danger can hardly be exaggerated. We must try laxatives and diuretics so long as the anasarca remains simple. The hot-air bath may be used cautiously, but there comes a time when it will increase the anasarca rather than diminish it. When acute hydrothorax or pleurisy with effusion occurs, the question of paracentesis presents itself, but is rejected not only by experience, but also on *à priori* grounds; both sides are involved at once, and the lungs mostly suffer as much as the pleuræ.

Sloughing of the cornea would probably be preventable in many cases by the simple expedient of keeping the eye shut, as recommended first by Trousseau.

DENGUE, OR DANDY FEVER.

By William Aitken, M.D.

Definition.—A febrile affection, *sui generis*, commencing suddenly, and associated from the commencement with severe pains in the large and small joints. About the third day a peculiar cutaneous eruption or efflorescence appears upon the palms of the hands, rapidly spreads over the whole body, and rarely continues visible beyond twenty-four hours. A distinct remission succeeds, but relapses are numerous, and the disease may thus persist about two months marked by prostration and cachexia, its course being characterised by intervals, or remissions, and the exacerbations marked by rheumatic or neuralgic-like phenomena.

Synonyms.—Scarlatina Rheumatica, Cock; Exanthesis Rosalia Arthrodynia, Cock; Dandy Fever, Natives of West Indies; Dunga Bouquet; Bucket; Epidemic Inflammatory Fever of Calcutta, Mellis; Eruptive Epidemic Fever of India; Toohutia, Natives of East Indies; Three-day Fever, Natives of East Indies; Rheumatic Fever with Gastric Irritation and Eruption, Furlonge; Eruptive Articular Fever; Eruptive Rheumatic Fever; Plantaria; Febris Exanthematica Articularis; Giraffe, on account of the stiff holding of the neck; Epidemic Anomalous Disease, Stedman; Peculiar Epidemic Fever; Colorado, on account of the red spots; Exanthesis Arthrosia; Stiff-necked Fever; Broken-wing Fever; Break-bone Fever.

History.—During many months previous to October 1824, there prevailed in Calcutta an epidemic fever so impartial in its attack that few remained untouched by the distemper in a population of nearly half a million of beings. Passing from the East Indies the disease was next heard of in 1827-8, amongst the islands of the Archipelago and in the Southern States of North America.

Between the months of September and January of these years it prevailed in the islands of St. Thomas' and Santa Cruz. Almost every individual in a population of 12,000 persons is reported to have suffered. (Stedman, Ed. Med. and Surg. Journ. Oct. 1828.) With few exceptions the disease spared no one of either sex, of any age, or complexion, or caste. The new-born infant, the young child, the aged, the weak, the robust, the rich, the poor, all were alike the objects of attack. Physicians, too, invariably became patients, and hence perhaps the details of symptoms in the epidemics of this disease are so extremely minute; and although the disease was a very painful one, it was not one dangerous to life. The attacks were invariably sudden. In families of ten or twelve persons (including servants), a half or even eight members would lie down at once (Furlonge, loc. cit. vol. xxxvii. 1832). Attention was therefore immediately arrested by epidemics of such a disease, and the general public, not less than medical men, were curious to learn the natural history of a disease with febrile characters so peculiar, and in results so unlike the epidemic or endemic fevers of tropical regions with which the physicians of the East and West Indies had been familiar. When the disease first attracted attention in Calcutta, it was generally believed that nothing of precisely the same nature had ever existed there before, unless the disease known to the native "conductors" of India by the name of "the Three-day Fever" were of the same nature. (Cavell, Trans. Phys. Soc. of Calcutta, vols. i. and ii.)

For the earliest accounts of this disease we are indebted to the physicians of the East Indies, and especially to Drs. James Mellis, Kennedy, Twining, Cavell, and J. Mouat; and the natural history of the fever given by them was subsequently corroborated, in all essential particulars, by the physicians of the Southern States of North America and those of the West Indian islands, more especially by Drs. Stedman, Cock, and Furlonge, in these islands, and by Dr. Dickson, of Charleston, in America. When the disease was first described by Dr. Mellis, he was disposed to regard it as "inflammatory fever;" but he found that such a name did not fully indicate the peculiar characters of the fever; nor could it be identified as exactly similar to the fever described by Cullen under the name of Synocha; nor to the fever described by Good under the name of Cauma; nor to the Febris acuta sanguinea of Hoffman. Many different opinions have accordingly been entertained regarding the nature of Dengue. By some it was considered as a rheumatic fever; by others as a remittent. Some regarded the complaint as measles; others as scarlatina. Some considered the fever synocha, with gastric irritation and an eruption associated with it, similar to that which in some constitutions follows fish-poisoning. Treatment therefore varied, alike in principle and in detail. But rather than subscribe to a definite name in any then existing nosology, the physicians who first described Dengue preferred comparing the phenomena of the peculiar cases they saw, as regarded their symptoms and sequelæ, with diseases of a similar kind; and so the first records of the history of this disease are peculiarly valuable when the speculations with which they abound are eliminated.

SYMPTOMS.—*Commencement, Development, Duration, Termination.*—The invasion of this disease as a rule was very sudden, and the progress of successive phenomena was rapid. Lassitude, drowsiness, heavy sensations in the eyes, frequent yawning, slight vertigo, a sense of coldness creeping down the back, or of numbness in the extremities which became cold, occasional rigors, pains in the head in most cases confined to the fore-part, or most severe there, acute pain in different parts of the body, sometimes in the larger muscles and joints, and occasionally in the smaller ones, such as the fingers and toes, are among the phenomena, one or other of which, or several combined together, suddenly expressed the commencement of the disease; and in the several epidemics whose histories have been recorded, some of these phenomena were more prominently marked than others. As a rule, however, the attacks were so sudden that no sensation of any deviation from the usual health indicated the approach of the malady. It often happened that people had a most violent attack, with severe headache and burning pains in the temples, within three hours after having boasted of their escape from the disorder. (Twining.) In general the patients woke out of their sleep with great pain in the head, loins, shoulders, arms, wrists, hips, thighs, and ankles, fingers or toes. (Mouat.) Acute pains in one or both knees, in the ankles, the wrists, or in all of these joints at once (though in general only one was at first affected), marked so suddenly the invasion of the disease, that the symptoms would first express themselves as the person walked along the street. (Stedman.) The motion of the joint was at first arrested, partly by stiffness, and partly from the pain caused by movement; and in cases where the symptoms commenced in the lower limbs the patient fell to the ground. The most usual mode of attack in the West Indian epidemics was expressed by a sudden stiffness, amounting to pain, in one of the fingers—generally the little finger. The stiffness increased, spreading rapidly over the whole hand and up the arm to the shoulder, so that in a few hours the fingers of both hands became so swollen, so stiff, and so painful, that all attempts to bend the joints were useless. At later periods in the various epidemics the approach of the fever was indicated at least a day before by anorexia, languor, listlessness, and a white tongue. A dry heat at the scrobiculus cordis was among the earliest symptoms noticed by Twining. The face was soon flushed—a phenomenon often observed by others before it attracted the attention of the patient. Intense headache followed upon the burning sensation in the forehead. The eyes became watery and the conjunctiva suffused. The whole countenance appeared bloated and swollen, the face assumed a scarlet hue, and the surface of the skin was everywhere flushed. The signs which then predominated were a chilliness extending over the whole frame, quickly followed by pain and weariness in the limbs, a general sensation of stiffness or soreness, with a heaviness over the eyes so excessive as to render the effort to open them painfully oppressive, and a headache so severe as to be beyond description. (Cavell.) Pressure over the eyes increased the pain, but light did not affect them. The eye-balls appear to the patient as too large for their sockets, as if ready to start from the head, and the pain in them so intense as to cause extreme distress. The expression of the eyes was ferrety. The lobes of the ears were likewise greatly pained. (Cock.)

The rapidity of the pulse, the aspect of the tongue, and the condition of the skin, each of which at first might not seem to be much influenced, yet rapidly passed from their normal condition.

The pulse, soon after the accession of the fever, was in most instances above one hundred per minute, becoming more frequent, full, hard, and strong, till it averaged about thirty or forty beats per minute above its normal rate, within six hours after the fever became expressed. Twining once observed it 140 in an adult whose usual pulse in health was eighty beats per minute. In some instances its force was so greatly increased that the temporal arteries were felt and seen to beat with violence. In children it was often so extremely rapid as to be indistinct and weak. Respiration was relatively extremely hurried; while determination of blood to the head was considerably increased, as indicated by bleeding at the nose, increasing redness of the face and eyes, confusion of thought, and sometimes delirium.

The tongue soon became of a scarlet colour at the sides, furred with a white or brown coat in the centre, so that in a few hours it appeared as if covered with a dense white paste, or with a thick dirty white coating, always moist, and associated with a disagreeably bitter taste in the mouth. The bowels were generally confined at first, while oppression at the præcordia, nausea, vomiting of viscid mucus and of bile were present from the commencement, and continued for some time. Irritability of the stomach was often indeed so very great that it retained anything with difficulty. The desire for food was inconstant. In some cases appetite was entirely gone; but not infrequently in children, the desire for food was increased. Thirst was not commensurate with the distress from other symptoms. Extreme prostration of strength became apparent at a very early period in the disease, and with rapid increase of pain in the loins, with not less severe pains in the muscles of the limbs, especially the legs, attended with a remarkable degree of anxiety and jactitation. The febrile anguish was extreme, with aching in the back of the neck. In short, suffering from pain was a leading feature in the accession and course of the attack of Dengue. The debility, the restlessness, and the general soreness rendered every position alike uneasy and intolerable, not less distressing to the patient than alarming to the spectator. (Twining.) Such was the excruciating nature of the pains, that few had fortitude sufficient to support them without complaint. (Cock.) These pains have been known to shift from one part of the extremities to another; sometimes attacking the patient in his knees, or in his toes, sometimes in one knee or toe, and sometimes in another; each new invasion of a part being accompanied with twitchings of the muscles of the part affected. So sudden were the attacks of pain in a fresh place, that a person might be calmly conversing with you when he would suddenly scream out from the severity of returning pain. (Cock.)

In some cases the headache and the pain in the back and loins were the most distressing symptoms, although not at all times equally severe during the first twenty-four hours. These pains would frequently subside a little to return in paroxysms with redoubled violence. It was, indeed, a disease of extreme severity as far as relates to the sufferings of the patient; but having regard to the result of the disease, as affecting life, and compared with its universal prevalence, it was a fever of unexampled benignity.

Such were the phenomena which, more or less severely expressed, marked the accession of an attack of Dengue; but towards the end of the first twenty-four hours the symptoms, which had gradually increased in severity, began to abate. The headache and flushing of the face somewhat subsided. The heat became more general and burning all over the extremities, to the relief of the head in some degree. With this remission of the pyrexia, the headache after the second day became still less, and the pains in the loins and other parts were attended with less jactitation; the character of the pain was changed to that of a dull aching kind, gradually leaving the fingers and ankles, and the toes last of all. During the first two nights there was little or no sleep, in consequence of the pain and febrile anguish; and although for several succeeding nights sleep was in most cases still interrupted by thirst and pains in the loins and legs, extending down to the toes and fingers, yet there was little or no disturbance of the intellect. Great prostration of strength; general debility of the whole system; weakness of the stomach, of the loins, of the limbs, of the knees, and of the joints generally; continued pain in several joints, large and small, sometimes limited to a finger only; œdematous swelling of the extremities; and general cachexia—denoted the exhausting and debilitating nature of the malady. The debility was so great that sudden death during the period of remission or of spurious convalescence was known to occur in some instances during the epidemic in Calcutta. The debility was not diminished by the sudden occurrence of perspiration, which during the early hours of the febrile accession had been suppressed; and although this return of perspiration was accompanied by warmth of the feet and a remission of all the more distressing symptoms, yet the prostration of strength was all the more apparent. The urine was now copious and pale-coloured; and the evacuations from the bowels, even if freely moved by remedies, were of a dark green colour, or even black, glutinous, scanty, and always offensive. During the three days following this remission, the patient, in the more severe cases, lay in a state of extreme languor, but irritable and restless; but as there are no records of temperature in this disease, it is not known if fever was entirely absent during the remission, although it is sometimes stated to have been so. It is rather to be presumed that the temperature did not fall to the standard of health; for although there was no acute suffering, yet the feelings were said to be very different from those of health. Thirst continued to prevail in some, and the desire for food did not return. The sense of taste seemed entirely lost; while in some cases little aphthous sores, on the inside of the lips and on the edge of the tongue, rendered attempts at eating painful in the extreme. (Stedman.) About the end of the third day the febrile phenomena again expressed themselves with even increased severity, compelling the patient to return to bed if he had ventured to leave it, deceived by the apparent but treacherous and spurious convalescence. With this febrile exacerbation, the cutaneous system exhibited remarkable and peculiar phenomena. With great turgidity of the skin, an eruption or rash appeared on different parts of the body. The appearance of this rash has been variously described :

(*a*) As an efflorescence, beginning at the palms of the hands, and gradually spreading over the whole body. In general appearance, this efflorescence differed considerably in different cases. In some it consisted of blotches of red-coloured skin, resembling, in appearance, something between measles and scarlatina. In some it was raised perceptibly above the surface of the skin, imparting a distinct feeling of roughness and elevation to the fingers passed over the eruption. In severer cases more obvious local swellings accompanied the efflorescence, and a distressing tingling of the skin resolved itself into an itching so intense, while the eruption disappeared, that the sufferers were almost driven to distraction. As a rule this efflorescent eruption remained only for about a single day, beginning to fade on the second day, and before the third morning it was generally entirely gone. Some degree of desquamation followed, generally in proportion to the intensity of the eruption. One instance is on record, in which a man eighty years of age had the eruption in so severe a form that the cuticle came off in flakes like pieces of parchment, leaving the surface of the body quite red. (Stedman.) The itching was extremely distressing, and the patient was in the utmost misery. In old men, the desquamation from the scrotum was attended with most intolerable itching, and in some of them the testicle became swollen to a great degree. In others, extensive abscesses formed beneath the skin, probably of the nature of the pyogenic fever of Tessier and Jenner, and popularly regarded as "the dregs of the fever." In the epidemic at St. Thomas's, two children, each about five or six months old, died from the irritation attending the desquamation of the cuticle. The true skin, so exposed, was red, raw, and exceedingly painful from the inflammation of its texture.

(*b*) About the third or fourth day an erythematous eruption appeared on the hands and feet (Cock), accompanied with swellings of those parts. This eruption gradually extended over the rest of the body, continuing for about thirty-six hours, when it faded, and the cuticle peeled off as in scarlet fever, leaving a considerable degree of soreness. The soles of the feet were sometimes rendered so sore, that walking, for many days after restoration to health, was attended with pain. The pains were apt to remain after the eruption had completely disappeared, and to become fixed in one or more of the joints, where they would remain for several months, with morning and evening exacerbations; the pains then were most severe. Sometimes the glands in the groins were swollen and painful. (Cock.)

(*c*) Dr. Furlonge regarded the eruption as altogether symptomatic of the gastric disturbance. He observed that its intensity and extent were proportional to that disturbance; that those who were "known dyspeptics" had the eruption more extensively than others; that literary or sedentary people, and those whose occupations were such as to derange the chylopoetic organs, suffered most from nausea and other symptoms of gastric disorder, and in them the eruption was always most extensive and marked. This eruption, he thought, resembled measles elevated with papulæ and wheals; a sort of hybrid between urticaria and rubeola, and of its nature similar to that which is known to attend the gastric disturbance from fish-poisoning. (Ed. Med. and Surg. Journ. p. 52, 1830.)

(*d*) Dr. Mellis regarded the eruption as similar to roseola or to the lichen simplex of Willan.

(*e*) Dr. Twining regarded it as resembling rubeola, while in some instances it resembled patches of an inflammatory appearance, in others a papillary, and in one or two instances that of a vesicular eruption. In many instances little more than a flush attended the febrile excitement.

(*f*) Mouat says of the eruption, that it is like erythema papulatum, or purpura simplex, not disappearing on pressure, and resembling roseola miliaris or lichen tropicus.

(*g*) In some cases there was said to have been no eruption (Cavell); but when the short persistence of the eruption is taken into account, it is not improbable that it may have been overlooked in such cases. In most cases indeed discoloration of the skin was evident from the first; and it seems to have been characteristic of Dengue that the eruption which attended it assumed many characters. In one case the eruption might be referred to the class papula, in a second to exanthema, in a third to vesicula, in a fourth to bullæ, and in a fifth to that of wheal. But whatever the form of the eruption, all the cases were attended during its continuance with very high excitement of the vascular system, which at last suddenly subsided, leaving the patient convalescent. The eruption came on suddenly and vanished suddenly, hence it may have been very often overlooked, or its characters might not have been observed, for it does not seem to have gone through any definite changes or marked stages. In some cases it appeared simultaneously with the febrile symptoms, in others not till twenty-four or thirty hours afterwards, and in two or three instances after all fever had apparently subsided. Wherever it appeared the colour of the eruption was similar, and the amount of the surface covered by the rash of various extent. In proportion to its early appearance it generally portended a further mitigation of the more distressing symptoms, and more especially if the efflorescence uniformly covered the whole body and the extremities. When the eruption was only partial on the body and less on the extremities, but increased about the chest, neck, and face, there was, for the most part, some increase of feverishness, more uneasiness and anxiety, with aggravation of headache. It remained persistent for a comparatively short time, and had for the most part considerably faded at the end of twenty-four hours after its first appearance, although it might continue obvious for two days. It was always attended with some degree of heat or itchiness, particularly when the minute exfoliations of the cuticle began to separate during the period of desquamation. It seemed to be chiefly in cases where the eruption remained persistent beyond two days that it assumed the aspect of urticaria, affecting more particularly the extremities; the fingers and toes, hands and feet being swollen, red, and affected with a distressing itching and burning, combined with febrile exacerbation and extreme debility for twenty-four or thirty-six hours. Associated with the eruption some patients had boils, and others had small acuminated vesicles with hardened bases. Children suffered most from urticaria, and in them large watery vesicles sometimes led to the formation of ulcers. In one case the sloughing was so deep, the ulcers so foul, and the fever so high, that, convulsions supervening, the case terminated by death. In most instances, though not in all, as in the epidemic at Suzuratte, the throat and fauces were so affected as to make deglutition painful. The secretions from the lungs and salivary glands were very copious, unhealthy in appearance, and distressing to the patients. The salivary glands were in some much swollen (the parotid as early as the fourth day), and the discharge of saliva in some instances amounted to ptyalism, although mercury had not been taken. It was also observed (Mouat) that very small doses of calomel frequently induced disagreeable ptyalism.

The disease was not considered to leave the patient till he had suffered from a second and even a third relapse or paroxysm of fever. These relapses were each at one time considered as separate attacks of fever; but the history of subsequent epidemics showed that these several paroxysms, each nearly equal in severity (although it was not noted whether the eruption repeated itself), combined to constitute one and the same attack of a fever marked by such peculiar remissions and exacerbations. There were also daily remissions observed by Dr. Mouat, there being two or even three paroxysms in twenty-four hours. Few recovered, under three months, from the debilitating effects of the attendant fever and the aching pains in the wrists, fingers, toes, and ankles. Tardy recovery, and the tendency to repeated relapses, were characteristic phenomena. In these periods of exacerbation the third days were decidedly "critical" (Kennedy); the pains in the limbs would abate, and the capability of taking food would return; but the furred tongue, the foul taste, and the disordered stomach generally continued for ten or twelve days after the final remission. The secondary prostration of strength evinced itself in proportion to the constitutional powers of the patient. Females in various periods of pregnancy went through the severer forms of the fever without any tendency to abortion.

Although the actual temperature of the body in cases of Dengue has not yet been recorded, there is reason to believe that the fever reaches its fastigium at a very early period—probably within three days; and during this period the surface of the body is subject to an irregular distribution of the blood, so that while the hands and feet are cold, the rest of the body, and particularly the head, may be intensely hot. During the height of the fever the head and eyes seem to indicate the greatest suffering, but the whole body is racked with pain, especially in the joints. Not an inch of the body from head to foot seems to have been exempt from suffering; hence the American name of "break-bone fever." In some cases the features, especially the eyelids, were swollen and distorted, associated in one or two instances with profuse ptyalism. But although the skin in cases of Dengue felt intensely hot to the touch of the observer, yet the patient experienced feelings of intense cold during all the different stages of the disease. When it prevailed as an epidemic at St. Thomas's, although the weather was extremely sultry, yet the patient felt the warmest coverings (of two or three blankets) scarcely enough.

ETIOLOGY AND PROPAGATION.—The origin of Dengue is unknown. The disease is said to have been first noticed in Rangoon about the end of May or beginning of June 1824; and on the 10th of June a large portion of the troops employed on the expedition under Sir Archibald Campbell, then at Rangoon, had been ordered out to attack the Burmese, and were thus exposed to incessant heavy rains for four-and-twenty hours.

The disease extended in various directions, not only to Calcutta, but to Chittagong in the south-eastern extremity of the Province of Bengal, and to Guzerat, in the Presidency of Madras. It was particularly severe in the large and populous towns of Benares, Patna, and Chunarghur, and prevailed generally from Buxar to Benares, Chunar, and Mirzapore.

In all of these places it seems to have become epidemic during or subsequent to the existence of heavy rains, associated with the close cloudy heat of sultry weather. Heat, moisture, and stagnation of air seem to have been always associated with the origin and transmission of the disease.

Stedman implies that the disease was imported into the free port of St. Thomas's, and the accounts of the disease generally show that it was propagated from place to place in the course or route of human intercourse. It travelled from Barbadoes to Jamaica. It raged in America and the East Indies. No disease indeed, with the exception of influenza, ever had so wide a diffusion. From St. Thomas's it proceeded upwards to Barbadoes. It seemed by the mode of attack as if something were applied which in a moment had the power of destroying the balance of health, and of producing a disease, the symptoms of which were so appalling at first sight.

Protracted debility, with long-continued pains in the limbs, were the invariable sequelæ of the fever. In several instances, tedious visceral disease, mainly of a sub-acute form, especially of the liver, with jaundice, were associated with the protracted duration of fever. Hæmorrhoidal affections sometimes preceded an attack of sub-acute hepatitis with slight jaundice. Returns of pains in the extreme joints of the fingers, distension of the abdomen, anorexia, slight tenderness of the belly, and thirst, betokened the advent of incipient visceral disease.

In a few cases the eyes were affected with Ophthalmia. (Mouat.)

Partial anchylosis is known to have occurred in the fingers of the hand. (Cock.)

DIAGNOSIS.—While the severe pains, on the one hand, caused the disease in some instances to be regarded as rheumatic, the paroxysms of the febrile attack and the intercurrent remissions, on the other hand, caused the disease to be considered as one of a malarious nature. But there was to be noticed the peculiar eruption already described, which, combined with the other characteristic phenomena, at once stamped the disease as one *sui generis*. The suddenness of the attack, the redness and the watering of the eyes, the acute pain in all the joints, rendered excruciating on the slightest touch, the scarlet or crimson efflorescence on the surface, its ephemeral duration, the disease sparing neither age, sex, nor habit of body, its seizing the acclimatised as well as those recently arrived, stamp Dengue at once as a disease different from the endemic remittent fevers of the countries where it has prevailed. The diagnostic differences from measles and scarlatina must be studied in connexion with the natural history of those diseases. Dengue attacked indiscriminately those who had suffered from scarlatina and those who had not. (Cavell.)

TREATMENT.—Amongst the natives of India Dr. Mouat had convincing proof that the disease was protracted and severe when no remedies were used. Prostration and severity were evinced in the great emaciation, the more extreme debility, the severer pains and the swollen extremities and the longer duration of the disease, in those cases left to run their own course.

Experience has proved that emetics and free eliminative remedies (especially purgation) insure an early freedom from fever. Time is no doubt an essential element in relation to the cure of the disease, which seems to run a specific course, and may be aided or retarded by remedies.

These remedies have been on the one hand (1) bleeding, on the other hand (2) purgation and eliminative medicines.

Twining did not resort to general blood-letting, but in several of the earlier cases in which the head was much affected, he sometimes applied a considerable number of leeches to the temples. A further observation of the progress of the epidemic and nature of the fever convinced him, however, that even leeches were by no means necessary, "as other cases with symptoms parallel in nature and severity were as speedily remedied without leeches." He considered that general bleeding was not adapted to the treatment of this fever. Cold affusion he also considered a hazardous remedy. The experience of Cavell also proved that depletion did not afford the relief expected of it. So also, Mouat records that bleeding did not cut short the disease, nor mitigate the symptoms. It added to the exhaustion of the patient; it impaired the powers of digestion; it induced vertigo during convalescence, with a tendency to fainting, distressing dreams, and bad nights. It was only of use in cases of local affection of the lungs, liver, or intestines.

With reference to purgation Twining's experience showed that a moderate dose of calomel, combined with an active dose of colocynth and scammony, and repeated every twenty-four hours, *till the evacuations were free, and of a more natural and healthy colour,* were the remedial agents he observed to be followed by the best effects. The use of purgatives in this way was indicated by the stools being of a dark-green colour; and with the disappearance of greenness from the stools, the symptoms were ameliorated. Calomel is never to be given alone. It is probable that calumba, rhubarb, and soda, combined in equal proportions, will equally answer as an alterative remedy. The eliminative action of purgation is to be obtained without the result of watery purgation; and so far as the use of purgatives is concerned, the only objection that is urged against them is the aggravation of the pain which they cause by the motion which their action entails. But the skill of the nurse and the physician must be combined to obviate this very secondary consideration. Eliminative remedies in an opposite direction were also shown to be of use. Emetics of tartar emetic and of ipecacuanha discharged large quantities of bile, relieving the pains of the head and of the limbs almost immediately. An emetic given at first always relieved the head and eased the pains; and this evacuation, followed by purgation, or even an open state of the bowels, tended greatly to facilitate recovery. A free, open state of the bowels is then best preserved by a dose (every two hours) of tartar emetic and sulphate of magnesia; or a compound, of jalap powder or of sulphate of magnesia, with infusion of senna. (Mouat.)

In thirty-six hours after the action of such remedies was obtained, the fever was subdued with less prostration of strength than by any other treatment; but if no bile followed the emetic, the symptoms were not relieved, and it was necessary to repeat it till the bile was discharged.

In the cases where ophthalmia was a consequence, leeches applied to the inner membrane of the eyelids sufficed for the cure, with free and repeated purgation.

After the bowels were freely opened, a light febrifuge mixture of sweet spirits of nitre, nitrate of potass, tartarized antimony, and colchicum, was attended with benefit, given every two hours, with an occasional effervescing draught, a pediluvium at bedtime, and ten to fifteen grains of Dover's powder. After all the acute symptoms had subsided, forty to sixty drops of the wine of colchicum, with twenty-five drops of laudanum, always insured a good night's rest, and thirty drops of antimonial wine were now and then added to the draught. (Furlonge.) Tonics (such as infusion of calumba, quinine, iron, or strychnia) ought to follow the free action of the bowels. Cordials, stimulants, and good diet, consistent with the habits of the individual patient, must also be thought of, and remedies of a local kind to allay the itchiness of the skin may also be of service; such as emulsions of almonds, with hydrochlorate of ammonia and corrosive sublimate judiciously combined and carefully applied.

ROSEOLA.

BY HERMANN BEIGEL, M.D. L.R.C.P. LOND.

DEFINITION.—Roseola is a disease characterised by the appearance on the skin of spots, separate from each other; of a roseate, scarlet, or dusky red hue; of minute size, like marks make by the point of a pin; but which may exist in such large numbers and so close together as to form large patches, of most varied shapes. These are not raised above the surface of the skin; are not communicated by contagion; and are generally unaccompanied by fever.

Willan is known to have described seven forms of Roseola, which number has been raised to twelve by Bateman, Rayer, and Wilson. Whoever wishes to take the form and hue of an irritation of the skin or any other part of the organism as a basis for classification, can, with great facility, introduce hundreds of new species into medical science. But whether science would gain anything by our calling a pneumonia, in which the inflamed part of the lung is round, pneumonia circularis, or, if triangular, pneumonia triangularis, is another question. Quite the same may be said in reference to roseola annulata and punctata; and if the words æstiva and autumnalis express anything, which may be taken as a basis for division, we might just as well find in the 365 days of the year material for a division into the same number of forms, not only of Roseola, but of any other disease. Classifications of this kind, which neither define correctly nor are of any utility, ought to be banished from medical science. Those who know the former unpractical, incorrect, and circumstantial classification of ulcers, and compare it with that of the present time, simplified and based upon correct observations,—admitting what influence nomenclature has on treatment,—will acknowledge the necessity of a judicious division in other spheres of medical science, and particularly in dermatology.

We may observe Roseola in the course of different, mostly feverish, diseases (Roseola symptomatica), or independent of diseases (Roseola idiopathica). The Symptomatic Roseola is a part of the disease in the course of which it occurs, and is therefore excluded from our consideration. We have now only to speak of Idiopathic Roseola, which rarely requires medical interference, but which gives us an important hint in reference to the pathology of skin-diseases in general.

Cause.—Anything which weakens or lowers the action of the nervous system, and this for a long time, may cause Roseola. Severe illnesses,—such as typhus or syphilis,—which change the vitality of the blood and nerves; bad nourishment, and general debility, are the common causes of R. symptomatica.

Idiopathic Roseola is brought about in a similar manner, but the action of the nervous system is weakened for a short time only. Not only errors of diet, but some particular kinds of food may produce Roseola. I know a lady who has a regular attack of Roseola, of seven to eight hours' duration, after eating strawberries, and this without any other disturbance; another lady who can voluntarily, produce Roseola by getting hot through dancing or other exertion and by drinking water afterwards. The eruption constantly appears in about half an hour, causes no other disturbance, and disappears after several hours. I attended a child who was troubled by hooping-cough. The milder attacks passed as usual, but the violent ones, during which the whole body of the little patient took a dusky hue, always brought on Roseola, which after a few minutes disappeared.

That sudden changes of temperature may produce Roseola is well known; and its appearance during dentition is remarkable. Sometimes it comes and goes so quickly, that a child, while teething, may exhibit five or six attacks during the course of one day. These attacks are usually unaccompanied by any pain. At the first appearance of the eruption, an anxious mother may send for the physician; but, so transient is its duration, that often, when the doctor comes, there is nothing to be seen. I am attending a family of two little boys and two daughters, and both of the boys have exhibited the phenomena of Roseola to a very high degree. The boys, who are strong and healthy, exhibit it at the commencement of every trifling indisposition, and this quite independently of dentition. The girls, who are twin-sisters, have not suffered. It is not rare, however, for Roseola, in some women, to precede every menstrual period.

It would appear, therefore, that Roseola depends immediately upon changes in the vitality of the blood and nerves; and we may be tolerably sure that if another Willan — endowed with Jussieu's capacity for observing nature—should appear, we should find Roseola, and many other skin diseases, ranging among the diseases of the nervous system.

Course.—Idiopathic Roseola appears almost feverless; yet feverish symptoms may exist, although they never reach a high degree. Headache, excited pulse, constipation, want of appetite, disturbed sleep, itching, may occur, but their occurrence is rather the exception than the rule. In the majority of cases Roseola idiopathica occurs suddenly, over a large surface, and without marked forerunners; it remains a few hours or days, and either disappears as suddenly as it came on, or turns into a more dusky hue, becomes at length pale, and a fortnight may expire before the last spots vanish. But it may happen that the rash disappears on one part of the body while new spots make their appearance on another.

In other cases a few little red dots may occasionally be discovered, and increase in number until the body is partially or totally covered with them. I never saw slowly developed Roseola disappear suddenly; but slowly as it came. The mucous membranes may participate in the eruption, but this is mostly observed in dentition, when these membranes are already affected.

That the same individual may repeatedly be subject to the eruption, is evident from the above statement.

Diagnosis.—Roseola bears the greatest resemblance to flea-bites, and to those partial inflammations of the skin which are caused by the stings of other insects. But bites and stings are distinguished from Roseola by the black central spot which, in an early stage, is clearly seen in almost all of them. Erythema Roseola differs in the fact that its confluent spots are raised above the level of the healthy skin. With measles and scarlet fever Roseola cannot be easily confounded; for besides the symptoms proper to those exanthemata, the skin, in the latter, is in a state of turgescence, and forms a red basis for the eruption, whereas in Roseola the skin between the spots is healthy. Its extremely mild course, independence of epidemic influences and character, and the absence of catarrhal phenomena, are sufficient to distinguish Roseola from measles.

Prognosis.—Idiopathic Roseola is a slight affection which never reaches a degree causing anxiety. Even in those cases where Roseola occurring during dentition is accompanied with fever, we scarcely have a right to refer the latter to the eruption, as it forms a symptom, which often occurs in dentition without being followed by Roseola or any other eruption.

Treatment.—Cases in which Roseola idiopathica requires medical interference seldom occur. The eruption comes and disappears without any disturbance of the general health. But if there exists a local irritation, which keeps up the eruption, or causes its recurrence, then, of course, it must be removed. Ascarides, which often cause Roseola, must be expelled; gentle laxatives will be applied against constipation, and impaired appetite improved by carbonized waters, soda, seltzer water, and by dilute mineral acids, particularly phosphoric acid. If during dentition the necessity arises to lance the gums, it will not be Roseola which necessitates this proceeding.

MEASLES.[1]

BY SYDNEY RINGER, M.D.

DEFINITION.—An acute febrile contagious disease, mostly occurring in epidemics. It generally attacks the patient but once, but sometimes again occurs after the interval of a few months, or many years. Relapses are very rarely met with. They may commence immediately, or three or four days after the fever of the first attack has declined.

Generally co-existing with epidemics of other diseases, it is especially related to hooping-cough. The one disease apparently predisposes to the other. Thus, often an epidemic of Measles precedes one of hooping-cough, or *vice versâ*. In the latter case, the paroxysmal cough may continue, or temporarily disappear during the existence of the Measles. It is stated that persons with pulmonary disease, such as bronchitis, are especially apt to catch the disease.

The period of incubation, in those cases in which the disease was produced by inoculation, was seven days.

SYNONYMS.—Measles; Flecken; Masern; Morbilli; Rubeola; Rougeole.

SYMPTOMS.—Measles is generally abrupt in its commencement, and is then ushered in by chilliness which may amount to rigors, or not uncommonly, in children, to convulsions; or, on the contrary, the invasion is so insidious that it is impossible to determine with accuracy the first day of attack. The disease, when established, is accompanied with a variable amount of prostration, but which is usually not extreme, and sometimes amounts only to a feeling of lassitude. The patients take voluntarily to bed, and are indisposed to either physical or mental exertion. They are fretful and irritable, in some cases only when disturbed; in others they are constantly restless, whining, and peevish—differences dependent on peculiarities inherited, or developed by bad education. The expression is vacant, and the powers of perception and reflection are much impaired. Delirium is in some cases present, always slight and usually limited to the night. The skin is hot and dry; the lips are parched, and in severe cases covered with sordes; the tongue, thickly coated, is mostly moist, and a few red papillæ may be often observed to project through the thick coating of fur. The appetite is much impaired, and the thirst often extreme. Vomiting not infrequent at the commencement, may be repeated and persistent, and then indicates a severe attack of the disease.

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[1] For much of the information contained in this article, the author is indebted to Willan's Miscellaneous Works, Dr. Armstrong's Practical Illustrations of Scarlet Fever, Measles, &c.; Trousseau's Clinical Lectures, Graves' Clinical Lectures, Hebra, Rilliet et Barthez' Maladies des Enfants, Journal für Kinderkrankheiten, Schmidt's Jahrbücher, Canstatt's Jahresbericht, Aitken's Practice of Medicine, Parkes on the Urine.

The bowels, in some cases confined throughout, are often relaxed, and are generally easily influenced by medicine. Diarrhœa not unfrequently first occurs at the period of the eruption. Usually from the very commencement the mucous membrane of eyes, nose, mouth, and respiratory tract suffers changes. The conjunctivæ are injected, and the eyes suffused with tears. There is generally some intolerance of light, and occasionally the patient complains of a sensation as of sand beneath the lids; the eyelids are swollen and red at the edges; there is often repeated sneezing, with at first a thin watery discharge from the nose; epistaxis not infrequently accompanies the coryza at its commencement. The mucous membrane of the mouth and throat is mottled with redness, and a feeling of weight and tension over the frontal sinuses may be complained of. There is generally some soreness of the throat, but without much swelling of that part. The cough is dry, hacking, and frequent, and there is a feeling of weight and oppression at the chest, at which part wheezing and rhonchal fremitus may often be felt. The voice is often hoarse; the respirations are hurried and shallow: the pulse is increased in frequency, but loses in force. The urine is scanty, and deposits an abundance of lithates on cooling. In rare instances severe pain in the abdomen has been noticed, with or without diarrhœa; pain and tenderness have been sufficient to lead to the idea of peritonitis, but they entirely disappear on the resolution of the rash.

Usually on the fourth day from the commencement of the disease the characteristic rash appears. First noticed on the forehead close to the scalp and on the chin, it from thence spreads over the face, trunk, and extremities, in the accomplishment of which it occupies a period varying from a few hours to two days. On the appearance of the rash the fever is stated to increase; it certainly does not diminish. The lacrymation of the eyes and injection of the conjunctivæ become more marked. There is slight swelling of the whole surface of the body (if the rash be abundant and general); this is especially noticeable in the face, and causes a variable amount of alteration of the features, so that in some cases the patient can scarcely be recognised. The feet and hands at the same time feel full and tense; deafness may be present, being due to the swelling of the mucous membrane of the Eustachian tube.

The cough at this time generally increases in severity, and is occasionally rather paroxysmal.

Examination of the lungs reveals sonorous, sibilant, and even a small amount of submucous rhonchus, the latter being most abundant at the bases of the lungs.

After the second or third day of the eruption the fever disappears, the temperature becomes normal, the pulse much less frequent, and the patient at once enters on the period of convalescence.

The period elapsing before perfect health is restored varies greatly; in some cases the recovery being rapid, in others prolonged to an indefinite period, this diversity being dependent on the previous state of health of the patient. Thus, if the health has been impaired by excesses of any kind, or by bad hygienic conditions of life; or if the patient be the subject of tuberculosis, scrofulosis, or syphilis, the recovery is often very greatly retarded, and more or less serious sequelæ often result. Further, other conditions being the same, recovery is more speedy in the young than in the old.

VARIETIES.—The severity of the disease varies greatly, and has thus led to its division into Morbilli mitiores and Morbilli graviores. Occasionally the disease is unaccompanied by any catarrhal symptoms, and is then termed Morbilli sine catarrho. This latter form is said not to protect the patient against another attack. Morbilli mitiores has already been described.

Morbilli graviores, Malignant Measles, or Black Measles, appears to have been far more common formerly than at present; still, cases do now and again present themselves.

Due either to individual peculiarity, or more commonly to a previously depraved state of health, or to epidemic causes, it may begin mildly, or the symptoms may be at once violent and severe. Usually the rash is but imperfectly developed, and partial in its distribution, of a livid, purplish, or even black colour, apt to disappear and again appear. There are marked prostration, great tremulousness, twitching of the muscles, delirium low and muttering in character, stupor or partial coma. The lips and teeth are covered with sordes, the tongue becomes dry and brown, the pulse is rapid and feeble, the respirations are hurried, and often the symptoms of congestion of the lungs are present. The extremities are cold, and petechial spots appear on various parts of the body.[1] The urine may contain blood, and bloody effusions are found in the various cavities and organs of the body. Death usually occurs early; should the patient rally, convalescence is much prolonged, and death not infrequently results from diarrhœa, bronchitis, or other of the sequelæ.

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[1] Dr. Veit (in Virchow's Archiv Bd. xiv. Heft. 142; reported in Canstatt, iv. p. 225, 1858) states that petechial eruption is in some cases dangerous, whilst in others the disease runs a favourable course. When serious, he says that the condition is brought about by previous excesses or irregularities of life, and that such cases are met with in all epidemics. He states that if the rash be at its commencement petechial, it is not an unfavourable sign; but that if it subsequently become so, if the rash change from winered to black, in form of round spots, extensive patches, or in streaks, then it is of almost fatal significance.

It is certain that not unfrequently small petechial spots may be met with on the upper part of the chest, and occasionally elsewhere, in cases which run a favourable course; but extensive and numerous petechiæ, especially if seated on the legs, are to be looked upon as of very serious import; though less so than if they occur in scarlet fever or small-pox, in which diseases their appearance warrants us in expecting a fatal termination; nevertheless, though this is the rule, cases of extensive petechial rash do occur, which run throughout a favourable course.

Rash.—Usually appearing on the fourth day, in some instances it is noticed as early as the first, or may be postponed to the seventh or eighth days of the disease.[1] Earliest seen on the forehead, close to the scalp, it from thence rapidly spreads to the rest of the face, then to the trunk, and lastly to the extremities, in the accomplishment of which it occupies, in some cases, but a few hours, but more commonly three days. On the first day chiefly limited to the face, it increases greatly in quantity over the trunk on the second, and is most abundant on the extremities on the third day of the eruption.

The development of the rash is best observed on the trunk. There are earliest seen minute red, not elevated, points, probably the papillæ of the skin reddened, and but little, if at all, enlarged. These rapidly develop, increase in breadth, and become elevated and acuminate in form, the diameter measuring at this stage about a line. In their further growth they increase chiefly laterally, and thus become flattened. The ultimate size they attain varies, although sometimes not advancing beyond the size mentioned when they are acuminate; they may become as large as a pea: in the former case they can be felt, and seem to be but slightly elevated; in the latter they feel hard, and even "shotty," though this is always much less marked than in the rash of small-pox.

The amount of the rash varies greatly. At first much scattered, fresh spots quickly appear on the previously unaffected skin. The spots are generally grouped, and often assume the form of crescentic or irregularly circular patches. Always at first discrete, they may become so numerous, that on enlarging laterally they coalesce and form patches of various size, sometimes sufficiently large to cover the chief part of the chest, or one or other of the extremities. These patches for the most part have their long axis across the trunk, they are always abruptly elevated, and the redness is sharply defined, though generally irregular in outline. Should the rash be very intense, the surface and redness of the patches are quite uniform; but mostly in the redness numerous small papillæ[2] can be seen, and even in the former case, on the decline of the rash, the papillæ become visible.

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[1] Of twenty-eight cases—

In 1, the rash appeared in twelve hours.
2, on the 1st day of the disease.
6, ,, 2d ,,
6, ,, 3d ,,
10, ,, 4th ,,
2, ,, 5th ,,
1, ,, 6th ,,

The commencement of the disease in these cases was dated from the very earliest symptoms noticed or complained of.

For some of these cases the author is indebted to Mr. Butt, and also to Mr. J. Bartlett, of Notting Hill.

[2] These are much larger than the punctiform elevations seen in scarlet fever.

Mostly abundant on the face, the quantity of rash developed becomes less the lower we descend, and the eruption is often only thinly scattered on the furthest extremities of the body.

The individual parts of the rash have a very short duration. Each spot, usually at its height in twelve hours, has often in twenty-four hours again subsided; so that no elevation remains, judging by both sight and touch—a mere red mottling being left. The rash declines in the order of its invasion. In rare instances, if the eruption be intense, small, clear, acuminate vesicles may be developed at the summit of the papillæ, or minute petechiæ may occur; these latter, usually not numerous, are perhaps most frequently met with on the neck, upper part of the chest, and the bend of the elbow.

Though generally described as of a rose colour, the rash appears to the author to be a mixture of red and yellow, the red greatly predominating at the beginning of the eruption; but as the spots enlarge and become flat, the redness declines, and the yellow tint becomes much more apparent.

It is of a brighter and more intensely red tint at those parts of the body which by exposure have become florid. It is thus well marked on the face and the hands. On the palms of the hands and the soles of the feet the papules generally look deeply situated, as if indeed they were placed beneath the cuticle.

By pressure the redness disappears, and a slight yellow discoloration of the skin remains.

After the rash has lost its elevation, a reddish, coppery discoloration is left. The redness (which is most marked, and remains longest on the face, where the vascularity of the skin is greatest) is always removable by pressure, quickly declines, and leaves a coppery discoloration. This coppery discoloration, on the other hand, is unaffected by pressure; occurring in spots a few lines in diameter, it is ill-defined in outline; sometimes of a uniform colour, but more generally punctiform; is proportionate to the previous intensity of the rash, and is consequently most marked on those parts of the body where the rash is most developed: hence it is well seen on the face, upper part of the trunk, but especially on the shoulders and over the scapular regions. It lasts a variable time, but has mostly disappeared by the tenth or twentieth day from the commencement of the disease.

The rash occasionally departs from the above description, in respect both of its nature and its course. Thus it may appear first on the trunk, and thence spread, sometimes involving, at others leaving free, the face; or the face may be the only part affected. In some cases it is very limited, the rest of the surface remaining free from rash. It is apt to appear earliest and to be most marked in the neighbourhood of sores or inflamed parts of the skin.

Sometimes, owing to a serious internal inflammation, mostly of the lungs, the rash suddenly recedes and may entirely disappear.

The desquamation is always slight, and is most marked on the face, or where the rash has been confluent: it is limited to the discoloured patches of the skin, and sometimes is only seen when the skin is tightened and viewed sideways; it begins usually on the sixth or eighth day of the disease, and continues eight to ten days.

The scales that are separated are usually very fine, often scarcely visible, unless dropped on a black ground. It is very rare for continuous pieces of the cuticle to be thrown off.

The elevation of the temperature, or fever, varies greatly in degree. The highest temperature reached in ordinary cases is usually 103° Fahr. If the temperature rises above this it indicates a severe, if it continues below this, a mild attack.

The temperature corresponds, in respect to its daily or other variations, to that of most fevers; thus it cannot assist us to make a correct diagnosis in doubtful cases.

The fever is stated to increase till the eruption and catarrhal symptoms reach their height.

As measured by the temperature, the disease has a very variable duration; in some cases declining on the fourth, or continuing to the eighth or tenth day of the disease.[1]

At the end of the disease, the temperature (*i.e.* fever) suddenly and greatly declines, but still usually rises slightly (to 100° or 101° Fahr.) on the one, and sometimes two days following. After this, it becomes normal, and continues so throughout convalescence; or it sinks for some few days below the point usual in health.[2]

The glands behind the jaw, down the neck, and in the groin become enlarged. The degree of enlargement behind the jaw is regulated by the amount of throat affection; that of the glands in the neck and groins, by the amount of rash present: hence the enlargement is greater in the neck than in the groins. The glands usually resume their proper size from the sixth to the tenth day of the disease.

The enlargement is rarely so great in any of these regions as it is in scarlet fever. In both diseases the enlargement is greater, the amount of irritation being the same in persons whose previous health has been bad, and thus the degree of enlargement serves as an indication for the treatment of the patient.

There may be no affection of the mucous membrane of the eyes, nose, throat, and lungs. Mostly, however, the whole of the tract suffers.

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[1] Of 14 cases—

2	ended on the	4th	day.
4	,,	5th	,,
4	,,	6th	,,
2	,,	7th	,,
1	,,	8th	,,
1	,,	9th	,,

It is right to state that in many of these cases the rash appeared before the fourth day, in several even on the first or second day, calculating from the day on which the very earliest symptoms appeared.

[2] The temperature in convalescence from all diseases is often below par, is easily depressed by exposure, and moreover is very slow to rise again after it.

On the conjunctiva, especially of the lids, is seen much fine capilliform and ramiform injection; the former declines as the rash fades, but the latter remains a few days longer.

The suffusion of the eyes usually continues some days after the injection has left, and in some cases may be noticed on the twentieth day of the disease.

Discharge from the nose, at first clear and watery, is one of the earliest symptoms observed. The mucous membrane of the alæ of the nose is red, and not unfrequently excoriated. On the decline of the rash, the inflammation also subsides; the discharge may cease immediately, but more commonly it becomes muco-purulent, and continues for a few days longer. If, however, the child has been badly nourished previous to the fever, or is the subject of the scrofulous diathesis, these symptoms are apt to be much more marked and of longer duration. The alæ of the nose are rather swollen. The mucous membrane, much excoriated, easily bleeds. The discharge is either thin and sanious, or thick and muco-purulent. This accumulates, dries, and blocks up the orifice. The upper lip may become much enlarged, hardened, and at first painful. The prolabium often cracks, bleeds, and scabs. These conditions may last from three weeks to as many months. Occasionally the discharge from the nose brings out an attack of eczema on the lip, which may spread over the rest of the face.

These appearances, being due to the previously impaired health of the child, become important indications in respect of the treatment.

The mucous membrane of the lips, gums, cheeks, soft and hard palate, usually reddened, in rare cases remains normal. The intensity of the redness varies, and it is not equally distributed, being usually most marked on the gums[1] and lips. On the cheeks and palate the redness, sometimes uniform, is mostly mottled. The gums are much reddened, rather swollen, and easily bleed. On the lips the redness, whilst uniform, is more intensely marked at places forming spots, mostly round, sharply defined, and especially noticeable in the neighbourhood of the frænum. Almost always on the gums, often on the lips, and occasionally on the cheeks and soft palate, are seen thin opaque white patches or films, looking as if the mucous membrane had been swept over with a solution of nitrate of silver; these vary in size, are often sharply defined, can be easily removed, and leave the mucous membrane entire, but red and sometimes bleeding. On the decline of the rash and fever, the inflammation of these parts subsides. The redness for the most part disappears, leaving only a coarse ramiform injection, best seen on the lips. The swelling of the gum subsides, but the whole mucous membrane retains for some time a swollen, spongy, and not unfrequently a glazed appearance. The white patches cease to enlarge, thin away, especially at their edges, and finally disappear; often, however, remaining for ten or fifteen days.

Occasionally ulceration is observed, usually slight—but sometimes severe—commencing close to the teeth (especially the incisors), appearing generally after the fever has subsided, but sometimes as late as the tenth or twelfth day of the disease. If slight, this usually heals in three or four days. It is noticed mostly in sickly children.

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[1] Especially that part corresponding to the incisor and canine teeth.

Aphthæ, and superficial, sharply cut ulcerations—usually small, round, or oval, but occasionally as large as a shilling, and irregular in outline, covered in some cases with an ash-grey, easily removable exudation, at others by a tough, smooth, leathery adherent membrane—are sometimes seen on the gums, lips, tongue, &c. They especially occur in certain epidemics. Of no danger, they heal in a week or ten days; the membranous form being more obstinate, lasting sometimes three weeks.

The tongue is rarely clean throughout; mostly thickly coated, moist, and with a few red prominent papillæ, it is sometimes dry and brown, the latter state indicating much depression, and being suggestive in respect to treatment. It begins to clear from the fourth to the eighth day of the disease. The fur is at first thrown off in the form of patches—leaving clear spots, sharply defined, oval or round, and about two to three lines in diameter. The rest of the tongue becomes clean by the eighth to the twelfth day, but still the patchy appearance remains, and can often be seen till the twelfth or fifteenth day of the disease. If any intercurrent disease supervenes, the tongue remains coated for a longer time.

The tonsils and mucous membrane of the pharynx, generally red and rather swollen, may be severely affected, and the tonsils may be so enlarged that they nearly meet. The mucous membrane, red, and at first dry, on the subsidence of the inflammation, becomes moist and covered with much muco-purulent matter. This ceases in a few days, leaving the mucous membrane spongy and sodden in appearance. Whilst the inflammation lasts there is usually some pain, especially on deglutition. This is usually not severe. The Eustachian tube may be involved, and there is then great pain, and difficulty of hearing. This is uncommon.

Vomiting, not common except at the very onset of the disease, may be persistent and continuous, the blandest food being immediately expelled. Sometimes the vomited matters are of a grass-green colour, usually at the same time there is diarrhœa with motions of a similar colour. Such vomiting only occurs in serious cases, and calls for a grave prognosis.

Diarrhœa, beginning usually at the time of the eruption, is not infrequent. Often severe, and sometimes bloody, it may cause death in young children. The colour of the motions varies; they are light-coloured, clayey, and sometimes green. The diarrhœa continues for a variable period, often after the fever has subsided, even to the tenth or twelfth day. The motions may continue clayey in colour after they have become less frequent and of greater consistence.

During the course of the disease, and for some time after, the bowels are very easily influenced by purgatives, a fact that should be borne in mind in the treatment of the disease.

The diarrhœa lasts longest in the weak and sickly.

The cough, at first hacking, frequent, and troublesome, in a few days becomes more annoying, violent, ringing, clanging, or often paroxysmal in character; sometimes so violent as to cause retching, and even vomiting; it usually greatly improves when the rash fades, and often disappears in a few days more. It may, however, continue for some time, retaining its paroxysmal character. The cough may first become paroxysmal as late as the eleventh or fourteenth day.

Expectoration, at first scanty, clear, and viscid, on the decline of the rash and the subsidence of the inflammation of the bronchial tubes, becomes muco-purulent, abundant, sometimes of a bright-green colour, and nummulated, the masses floating in a clear, thin watery fluid. It usually quickly lessens in quantity, and mostly disappears by the tenth or twelth day of the disease.

Urine, scanty, and on cooling depositing an abundance of lithates, has generally, during the fever, a peculiar yellow colour. Albumen in small quantities is frequently, blood less commonly, found during the fever days. Both the urine and breath have a peculiar odour. This, in the author's experience, is only met with in children, in whom it can be detected in all acute febrile diseases, especially when the mucous membrane of the mouth is affected.

Complications and Sequelæ are either the usual anatomical lesions, so increased in severity, or continued after the fever has declined, as to become of serious importance; or intercurrent diseases not usually attendant on Measles. Very various in nature, and often of great severity, they not unfrequently terminate in death.

They vary in different epidemics, and are, to some extent, influenced by the season of the year. Pulmonary affections are more common in winter than summer. Some are moreover dependent on the previous depraved health of the patient, such as the gangrenous inflammation.

Convulsions, not uncommon in children at the very commencement of the attack, are usually without danger. They may however be repeated, and terminate fatally. Occurring by no means commonly, at a later period of the disease, they are then usually repeated, and accompanied with some severe internal inflammation, mostly of the lungs. At this period they are generally fatal.

Laryngitis, commencing with slight cough, soon followed by much oppression at the chest, hoarse cough, noisy and stridulous breathing, occurs before the eruption, or with a recession of the same. It may be croupous or diphtheritic.

It mostly disappears on the evolution of the rash, and, if not then, on the decline of the fever.

Chronic inflammation of the larynx is commonly met with, and generally proves obstinate. It disappears only to return again on slight exposure to cold. The voice and cough are hoarse, and sometimes at the same time there is deafness in one or both ears. When the latter occurs the tonsils are usually enlarged, and at the same time the patient snores much during sleep. This affection may continue to recur for several years.

Chronic inflammation of the bronchial tubes, with or without the last-mentioned sequelæ, may follow Measles. The cough is hollow, ringing, hacking, or sometimes paroxysmal, and is easily aggravated by exposure to cold. With or without expectoration, but accompanied usually by some oppression of breathing, it may continue with remissions, or for a time disappear, but may, for years, return on exposure to slight exciting causes.

Capillary bronchitis may occur either during or subsequent to the decline of the fever. It is almost always fatal in young children under two years of age.

Pneumonia, simple or lobular, occurs both as a complication or as one of the sequelæ.

The symptoms of lobar pneumonia are mostly well marked and characteristic; they may, however, be masked or take on the character of those of typhoid fever.

Lobular pneumonia, by no means uncommon, is often difficult to diagnose. The fever is high, the respirations are much hurried, the nares act strongly with respiration. Dulness on percussion of the chest is absent always at the commencement, and sometimes throughout the disease. There is heard, irregularly scattered over the whole of both lungs, fine crepitation—occurring with both inspiration and expiration—but in children this crepitation is by no means easy to distinguish from the subcrepitant rhonchus of bronchitis.

In bronchitis the rhonchus is at first limited to, and always most marked at, the bases of the lungs. It is usually larger and more evenly distributed than the crepitation of lobular pneumonia, which is generally limited to different parts of the lungs. In bronchitis the fever is mostly less severe, and there is greater lividity of the lips, with less disturbance of the respiration. But, though the above differences often hold good, yet cases occur in which it is often impossible to make the diagnosis. Its influence on the rash is greater than that of bronchitis, the fever being higher.

Broncho-pneumonia is by no means uncommon both as a complication or one of the sequelæ of this disease. The disease begins as ordinary bronchitis; this increases in severity; abundance of loose, subcrepitant rhonchus is heard over the chief part of the lungs, at first limited to, and always most abundant at the bases. This is at first moist and distant; but as the consolidation advances, it sounds very superficial, ringing, dry, and high-pitched. This change in the character of the rhonchus is usually the only sign by which it can be told that the bronchitis has passed into broncho-pneumonia. For mostly all dulness is absent; nay, there may be, and at first generally is, increased resonance, whilst the respiratory sound is merely harsh, or completely covered by the rhonchus. If the consolidated portions coalesce, dulness may be developed, and tubular breathing caught, but this only happens in rare and very severe cases.

In this affection the fever is very high, the nares dilate greatly during respiration, the face is livid, the respiration very greatly laboured, the jugular vein often full, and the expectoration, where it occurs, is bronchitic in character.[1] The child very frequently, perhaps generally, sinks; the surface becomes cool; the pulse weak, feeble, and fluttering; the respiration superficial: the expired air cool; sordes collect on the teeth and gums; delirium of a low muttering character is present. There is a muscular trembling, picking of the bed-clothes, and after a period varying from six to twelve days the child dies.

It mostly occurs in children from two to six years of age, and is very frequently fatal. Its duration is said to be two to four weeks, but the child either dies before, or the fever subsides on the eighth or tenth day. Rhonchus may continue to be heard some time longer.

Beginning as simple bronchitis, the inflammation spreads laterally from the fine bronchial tubes, producing minute red spots or streaks. Red and then grey hepatization soon follows, and little bodies varying in size from a millet seed to a pea are formed.

Ulceration may begin in the mucous membrane of the bronchial tubes of the hepatized tissue, and spread laterally, till small cavities, filled with a greyish tenacious fluid, are formed. A bright red line in the hepatized portion, and bounding the cavity, may be seen. These hepatized portions of lung may be so numerous that as they enlarge laterally they coalesce, and thus much of the lung may become completely consolidated, and such portion is sometimes riddled with small cavities.

When in the state of red hepatization, the little bodies mentioned look not unlike tubercle, but they are most abundant at the base, less sharply defined, stand out less prominently, are less dense, and are at the same time more granular-looking.

Gangrenous inflammation of the mouth usually begins insidiously: it occurs sometimes during, but mostly after, the fever. There is at first slight excoriation, the inflammation then quickly spreads, the parts become much swollen, and the eye may be closed. The tissues become hard, red, and painful. The surface is sometimes at first covered with a dirty, fœtid, grey exudation. The tissues slough, the cheek may be perforated, the gums destroyed, and the teeth drop out. Both cheeks may be affected. The affection is only met with in children, who almost always die.[2] Similar inflammation occurs sometimes in the vulva.

Acute tuberculosis or chronic phthisis may occur during the course of the disease, but it usually first gives evidence of its existence after the fever has declined. Acute tuberculosis follows Measles more frequently than any other of the acute specific diseases, hooping-cough being perhaps excepted.

Bleeding at the nose in rare cases may be so great as to endanger life.

Inflammation of the ear may at first escape notice. The child cries greatly, and apparently without cause, and puts its hands to its ears.

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[1] Children under twelve rarely expectorate.

[2] See Cancrum Oris. Vol. III.

There may be some redness and swelling of the meatus. On the third or fourth day a muco-purulent discharge occurs.

Gangrene of the lung, diphtheritis of the labia, acute Bright's disease with anasarca, and some other complications, may occur.

It may be added that the complications occurring during the eruptive period are usually without danger.

DIAGNOSIS.—This is made chiefly from the rash, and the co-existing coryza.

As the rash in the different stages somewhat simulates that of scarlet fever, roseola, small-pox, typhus fever, and syphilitic roseola, the distinction between these rashes must be mentioned.[1]

Scarlet Fever.—The rash appears on the second day of the disease. It is very generally diffused. In Measles the papillæ are often widely scattered. The elevations always present in scarlet fever are very small (punctiform); those in Measles much larger (papilliform). Where the rash of the Measles forms patches, the elevations seen in the general redness are larger than those of scarlet fever; the edges of these patches in Measles are sharply defined and abruptly elevated; in scarlet fever, ill-defined and not at all elevated.

Occasionally in scarlet fever there are a few prominent elevations on the arms and legs; these may be mistaken for the papillæ of Measles, but the rash on the rest of the body is sufficient to establish its true nature.[2] The tint of redness in most cases is different in the two eruptions. Coryza is not noticed in scarlet fever, though the conjunctivæ are almost always injected.

Roseola Æstiva may have a rash not unlike that of Measles. This rash in appearance is intermediate between Measles and scarlet fever, but generally more closely resembles the latter. Thus, the rash is generally much diffused and ill-defined, the elevation is slight, coryza is absent, and the fever is trifling. It frequently occurs many times in the same patient, and often at the same period of the year.

Small Pox.—This disease in some respects corresponds to Measles. In both there are catarrhal symptoms. In both small red points, beginning on the face and then spreading over the rest of the body, are met with. In small-pox the disease begins abruptly; in Measles not infrequently insidiously. In small-pox there is often severe pain in the back and head, with much vomiting. These symptoms are most generally absent in Measles. The rash of small-pox is from its commencement hard and "shotty," quickly becomes crowned with an umbilicated vesicle. Moreover, the fever greatly declines or even disappears temporarily, when the rash is fully out, in both respects differing from Measles. In small-pox, if unmodified, a characteristic smell is present. The rash appears on the third day in small-pox, usually on the fourth day in Measles, but the departure from this rule in Measles is so frequent, that no great stress can be laid on this distinction.

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[1] The rash of Measles is often, at its commencement, compared to that of flea-bites, but the differences between them are always well marked, and by the most superficial examination they can be distinguished. Flea-bites take on two different forms. In the simpler there is no elevation, but a central point of redness, not removable by pressure, surrounded by a small ring of a less intense colour; this latter redness disappears on pressure. In another form there is a round firm swelling, varying in size, often as large as a pea; this itches greatly. It has all the characters of the wheals found in urticaria.

[2] It is stated that the two fevers may co-exist, and that these rashes may be mixed. No such case has come under the author's notice.

Typhus Fever.—The mottling left after a severe attack of Measles is often extremely like the petechiæ of typhus fever, but the early appearance of the rash of Measles will in all cases be sufficient to ensure a correct diagnosis. The rash of typhus fever, moreover, appears first on the wrists.

The mottling left by the rash of Measles is in all respects like the mottling seen in syphilitic roseola, but the rashes are different at their commencement, and the mottling left by Measles is most marked on the shoulders and over the scapulæ, the mottling of syphilitic roseola is generally absent from these regions, and is most marked on the trunk. Moreover, the general symptoms are sufficiently different to render a mistake impossible.[1]

Occasionally copaiba, morphia, and certain fish, produce a rash which in some instances it is impossible to distinguish from that of Measles. The rash, however, produced by these causes, is often very irregular in its distribution, and lasts a much longer time than that of Measles, if the medicine be continued. Occasionally there are, mixed with this rash, wheals of urticaria; when such is the case, there is much itching.

PROGNOSIS.—Usually a mild disease, the prognosis is very favourable.

The degree of danger is dependent on the previous health of the patient, on the time of year at which the disease occurs, and on the nature of the epidemic.

If the health has been bad, the prognosis is more serious, but even in such cases the result is mostly favourable.

If the patient be tubercular, the prognosis becomes grave. Cold and damp weather, favouring the development of pulmonary affections, increases the danger of the disease.

The severity and the nature of the complications occurring during different epidemics, vary greatly. Yet, in the worst epidemics, the majority of cases recover.

TREATMENT.—Not possessing any medicine capable of shortening the duration of the disease, it must be our object, in treatment, to conduct the fever to a favourable termination, and to ward off any intercurrent disease.[2]

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[1] The author believes that Dr. Hare was the first to point out this resemblance between the rash of Measles and that of syphilitic roseola.

[2] The remarks made respecting the treatment of Measles, of course apply for the most part to all febrile diseases, and especially to those of long duration.

The patient must be confined to bed. Too much light should be excluded, for strong light is annoying to all febrile patients, and in Measles causes some pain in the inflamed conjunctivæ.

The room should be well ventilated, and all excreta and dirty linen immediately removed. The patient must be most carefully protected from draughts. The sense of heat and dryness of the body, sometimes most distressing to the patient, can be much alleviated by washing the surface with soap and tepid water; too great exposure being avoided by one part of the body being cleansed, dried, and covered, before the rest is exposed. The feeling of tension of the hands and feet can be relieved by rubbing these parts with some firm greasy matter, such as suet.

All sources of annoyance or irritation, all noises, should be avoided, and thus sleep is promoted, a condition which most materially affects the welfare of the patient—sleep lessening the fever and increasing the appetite. Food, light and nutritious, such as arrow-root, gruel, good beef-tea, milk, chicken or veal broth, jellies, &c., should be given at the usual meal hours. The quantity should be moderate, great care being taken that the digestion be not impaired by too large ingestion of food. Should the patient be very weak, the food must be administered in small quantities and at short intervals. There is no stimulant so important as food. The prospect of recovery in all acute febrile diseases is very greatly, if not mainly, dependent on the power possessed by the patient of digesting and assimilating food.

If there be often-repeated sickness,[1] food of the very blandest nature, pounded raw meat,[2] Liebig's beef-tea,[3] uncooked white of egg diluted with water, or barley water, should be given in small quantities, and be very frequently repeated.

Thirst must be assuaged, and thus the restlessness of the patient allayed, by drinks, regulated with respect both to quantity and quality. Large draughts should be prohibited, as they tend to impair the digestion, and cause diarrhœa; small quantities, swallowed slowly, or ice to suck, are sufficient to allay thirst, and they also prove grateful to the patient.

The patient, however, must be allowed to take larger quantities of fluids than in health, as an increased quantity is required by the system during the existence of fever.

Acid or acid and bitter drinks are generally found to lessen thirst to a greater degree than mere water, and are moreover grateful to the patient. "Imperial drink," lemonade, with very little sugar, raspberry vinegar, or weak infusion of cascarilla, with a few drops of hydrochloric or nitric acid, are all—especially the latter—useful.

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[1] A very unfavourable symptom, for sickness itself very greatly prostrates the patient, and by the vomiting the nutritious matters are returned; nay, more, the vomiting is due to a condition of the mucous membrane of the alimentary tract most unfavourable to digestion.

[2] Before pounding the meat all fat and tendon should be removed.

[3] Liebig's beef-tea is made by cutting one pound of beef very small, pouring on this one pint of water, to which has been added thirty minims of hydrochloric acid and two scruples of common salt, and allowing this to stand three hours. It should then be strained and strongly expressed: it is then fit for use.

Stimulants are administered to support the strength of the patient. This they do in a great measure by promoting digestion, and by also directly increasing the force of the heart's action; for in fever-patients the pulse, under the influence of alcohol, most certainly gains in force.

The administration and quantity of stimulants given must thus be regulated by the condition of the patient. Guidance in this respect can be derived from the previous history of the patient, the existing state of the patient, and the nature of the epidemic.

If the patient, previous to the fever, has been in an unhealthy state from any cause, such as overwork, excesses of any kind, or is the subject of scrofulosis or tuberculosis, in whom the fever always tends to become typhoid in character, and to leave one or more of its sequelæ[1] behind, stimulants may with benefit be given early in moderate quantities; and at the same time the patient should be carefully watched, so that their quantity may be increased in anticipation of any great amount of prostration.

Again, in some epidemics the disease is violent, and apt to become typhoid. Patients met with in such epidemics may have stimulants given them early, with benefit. But the chief information respecting the administration of stimulants, and the quantity of these to be given, is derived from the then existing state of the patient. If the disease be progressing favourably (the previous health having been good), stimulants had better be altogether abstained from. But if the patient become at all prostrate, the tongue dry, the pulse small and frequent, stimulants must be given freely and often. It should be the anxious endeavour of the physician to anticipate such a state, and prevent it by the early use of alcohol.[2]

In reference to the amount to be given, no exact rules can be laid down. Experience at the bedside alone can teach this. It may, however, be stated that young children bear stimulants well in disease.

It is a matter of but little importance in what form the stimulant is given, provided it is sound in quality. The patient's taste should be consulted. Thus, brandy, gin, whisky, port or sherry wine, bitter or draught beer, may be given.[3]

Only one kind of stimulant should be administered at the same time, though it may be frequently changed at the request of the patient.

At the commencement of the disease, if vomiting has not occurred, an emetic can be often given with benefit, and the bowels, if confined, should be opened by a mild purgative, it always being borne in mind that in Measles the intestines are usually very easily influenced by purgatives. Acetate of potash, acetate of ammonia, and nitric ether may be given in suitable quantities every three or four hours; and if the cough be troublesome, some ipecacuanha wine may be added. If the skin be hot, the pulse hard, and incompressible, aconite, given hourly, will be of much service.

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[1] The probability that the various anatomical lesions met with in Measles will become chronic, is determined by two conditions—first, by idiosyncrasies, inherited or acquired—thus patients who have had many attacks of bronchitis, will be very liable to have some chronic bronchitis left behind;—and secondly, and chiefly, by the previously impaired health of the patient.

[2] Whilst in such cases stimulants should be freely given, it must not be forgotten to give the patient food in small quantities, frequently repeated; for food is the best and most natural stimulant.

[3] The patients will sometimes take stimulants in this form when they refuse them in all others.

Opiates are but rarely required; sleep should be obtained by removing, as far as possible, those conditions that are liable to render the patient restless.

Bleeding at the nose, if severe, should at once be stopped. This can be accomplished by the injection of cold water, or the breathing up of astringents, in fine powder, into the nose; should these fail, plugging becomes necessary.

For the convulsions that occur, often but little is required, and but little can be done—those occurring early quickly disappearing; those occurring late almost always ending in death.

In the treatment of laryngitis, Trousseau warns against the application of leeches, and adopts Graves' recommendation of a sponge wrung out of boiling water, and applied over the larynx.

If suffocative catarrh supervene, or a serious internal inflammation cause the recession of the rash with much prostration and possibly with coma, a warm bath, to which two tablespoonfuls of mustard have been added, may be used with benefit. The child should be kept in the bath till the arms of the nurse holding the child tingle and smart.

Gangrenous inflammation is best treated locally, by the application of strong hydrochloric or nitric acid, and generally by the free use of stimulants, with sesqui-carbonate of ammonia and tonics.

If there be much inflammation of the fauces (but this is not usual), linseed poultices to the throat, and the inhalation of steam, or ice to suck, will be found of benefit. On the subsidence of the inflammation, if the formation of muco-purulent matter continue, adults can use astringent gargles. It must be borne in mind that children under eight years of age cannot gargle, and thus the fluid should be injected into the throat with a syringe. There is no fear of choking, provided the stream of the injected fluid be not too great.

Inflammation of the lungs, when occurring in children, requires active stimulant treatment. Bleeding in these cases can never be borne.

The chronic sequelæ being dependent on the impaired health of the patient, are best treated by those means and medicines that promote the general health of the patient, and also, when possible, by local applications. The child should be warmly clothed, with flannel next the skin. Much of the day should be spent in the open air and in direct sunlight, unless the patient be too young, or the sun's heat too powerful. Hours of rest should be long, and sleep may be indulged in with advantage for a short time during the afternoon.[1] Simple healthy food, at regular hours, with a very moderate allowance of stimulants, or even without any, should be given. Sea-bathing or cold-sponging will be found to be of the very greatest advantage for the cure of these sequelæ. The sea-bath should be taken about three hours after breakfast. If the patient be very weak, a very short stay in the water of one to two minutes at most should be allowed. The sea should be smooth, and if the weather be wet or stormy or cold, a tepid sea-bath is preferable to open-air bathing. Children should be coaxed, not dragged, into the water, as baths fail to act as a tonic, and produce much depression in persons labouring under strong mental emotions.

The cold sponge-bath may be used before breakfast. If the patient be very weak, and the weather very cold, a little warm water should be added. The time the sponging should be continued depends on the condition of the patient; the weaker the subject the shorter the time; thus one to two minutes is in many cases as much as can be borne, for if continued too long, in such cases no tonic effect follows, but depression, which generally lasts during the rest of the day. Very young children can be much benefited by cold-sponging, even during the winter months, if the bath be properly used. The sponging should not be continued longer than a minute, and if the weather and water be very cold, the child's feet should be placed in warm water, and the bath administered before a good fire; by these means reaction is promoted. Salt may be added to the bath with benefit.

Under such treatment all the sequelæ greatly diminish, nor need there be any apprehension that the chronic bronchitis should be aggravated; for if the baths be administered in the manner described above, this disease almost invariably greatly improves.

Of medicines, iron, quinine, and especially cod-liver oil, are always very beneficial. The latter should be administered after food, for at this time the bile is poured out in largest quantity into the intestinal canal, and it is found that animal membranes moistened with bile allow fats to pass through.

The chronic discharge from the ears, nose, and vagina is usually easily arrested by local applications. The affected part should be washed frequently (in proportion to the amount of discharge) during the day with tepid water, and injections of lead lotion, or of solutions of alum (ʒj. to ℈j.), or sulphate of zinc (gr. iv. to ℥j.), will generally promote the cure.[2]

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[1] Every care should be taken, during convalescence, to ensure sound sleep. The supper should be light, and be taken one or two hours before going to bed. No stimulants should be taken during the evening, unless the patient be very weak. If baths cause restlessness, they must be discontinued for a few days, or the patient should be directed to remain in the water for a shorter time. During convalescence the patient is in much the same condition as a child. It is a period, if not of growth, of great repair—a condition analogous to growth. The appetite, digestion, and assimilation are greatly influenced by sleep. This influence is well seen in ulcers on the surface of the body. After a restless night these are painful, throbbing, inflamed, and swollen, and apt to spread, whilst after a refreshing sleep they have a much healthier appearance.

[2] In the author's experience lead lotions are preferable to the others.

The chronic inflammation of the larynx, or of the bronchial tubes, is most quickly mitigated and generally cured by inhalation of steam with ♏x. to ♏xx. of tr. iod. poured on the water, from which the steam is given off. Creasote may be sometimes used, but does not give such good results as the iodine. The inhalation should be used night and morning for about ten minutes.[1] The chronic diarrhœa, which occasionally follows Measles, should be treated by a carefully regulated diet, and the occasional use of hyd. c. creta. If the diarrhœa be exhausting, it can be generally temporarily stopped by an injection of starch, of the consistence of cream, and about two ounces in quantity; to this some laudanum or one of the metallic astringents may be added.

Raw pounded meat sometimes has a beneficial influence on the diarrhœa.

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[1] The cough is greatly influenced by change of climate. If the cough be easy and the expectoration scanty, a warm moist climate, but if the expectoration be abundant, a warm dry climate is beneficial.

PAROTITIS.

By Sydney Ringer, M.D.

Definition.—An acute febrile disease, characterised by an anatomical lesion situated in one or both parotid glands, which runs a short course, and almost invariably terminates favourably.

Synonyms.—Parotitis ; Cynanche parotidea ; Ziegenpeter (Germ.) ; Parotide, Parotidite (Fr.); Mumps (Engl.).

Symptoms.—Beginning abruptly—rarely with rigors, more commonly with a feeling of chilliness, with or without vomiting, pain in the head, back and limbs—the disease in its course is accompanied by the symptoms common to all febrile diseases. The face is sometimes flushed, the lips may be dry ; impairment of strength, variable in degree, is generally slight, and sometimes absent; the tongue is furred, but usually moist; the appetite, in some cases natural throughout, is in most impaired, and may be *nil.* The patient usually complains of thirst, and the bowels are often confined.

The pulse and respiration are increased in frequency, the former often greatly so, especially in children; it also sometimes gains in force ; the urine is scanty and high-coloured ; the temperature of the body is raised, but this elevation varies much in degree.[1] Usually at the very commencement of the disease, but occasionally postponed for twelve, twenty-four, or even thirty-six hours, the affection in one or other parotid gland, sometimes in both, manifests itself by pain, followed in a few hours by swelling of the glands and stiffness of the jaws.

The pain and swelling first appear immediately beneath the ear, and posterior to the ramus of the jaw, and from this part spread in all directions, upwards to the face, downwards and backwards in the neck. On the face the swelling appears earliest on that part immediately in front of the lobe of the ear, and then quickly extends upwards to the zygomatic arch, and forwards involving a variable extent of the face. The swelling disappears in the inverse order of its invasion. It is at first flat, but soon becomes more prominent, and is usually most marked anterior to the lobe of the ear. Firm and elastic to the feel, it is generally tolerably well defined, but does not pit on pressure;

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[1] In one case the highest temperature reached was $101\frac{3}{5}$; in another, however, it rose to $103\frac{4}{5}$.

and, whilst the skin over the enlargement is mostly natural in colour, it is in some cases mottled with a slight red blush, and is in rare cases of a bright scarlet hue. The redness disappears on pressure, but quickly returns on the pressure being removed. The degree of enlargement varies; whilst in some cases apparent only behind the ramus of the jaw, or even so slight that it easily escapes notice, it is more usually co-extensive with the parotid gland, and occasionally extends far beyond this, involving a large part of the face and neck, and, in some rare cases, reaching to the upper part of the chest, giving to the head and neck a pyramidal shape. The swelling increases from three to six days, then usually remains stationary for twenty-four or forty-eight hours, after this rapidly declines, and often has entirely disappeared by the eighth or twelfth day from the commencement of the attack; the redness subsides much earlier, and is occasionally followed by a superficial desquamation of the cuticle. Not unfrequently one or both submaxillary glands are also involved, in which cases the swelling extends along the body of the jaw, reaching nearly to the symphysis.

The pain and tenderness, dull and aching in character, vary greatly in degree; sometimes they are only complained of on movement of the jaw, and are then seated beneath the ear, and behind the ramus of the jaw—a fact easily accounted for when it is remembered that this part of the enlargement is most affected by the movement—in other cases, however, the pain is constant and severe, and occasionally extends beyond the limits of the apparently affected tissues, reaching even to the chest and shoulder.

The pain, more severe in adults than in children, usually lasts only during the time the swelling is increasing; on the other hand, tenderness on pressure, which is always present, continues for some days longer, and is longest observed in the part first affected. The jaw is generally fixed, and the mouth slightly open; moreover, its movements are limited, or entirely prevented, the degree of impediment being proportionate to the amount of pain and swelling present. Consequent on the impediment in the movement of the jaw just mentioned, the speech is affected, and mastication can be but most imperfectly and painfully performed; and when, as is sometimes the case, the tonsils are enlarged and inflamed, and the swelling extends even to the cellular tissue of the pharynx, deglutition also becomes difficult, and danger of suffocation, in very rare instances, is imminent. The mucous membrane of the mouth is unaffected, and the salivary secretion, in some cases diminished, in a very small number increased, is usually natural in both quality and quantity.

Occasionally only one parotid gland is affected; in most cases, however, both suffer. The left side is most frequently the first involved, and the pain and swelling in it precede that in the right for a period varying from twelve to thirty-six or forty-eight hours. The side first attacked suffers the most severely. It is stated that, in rare instances, the gland has suppurated.

Other organs besides the parotid and submaxillary may be affected. In many the testicles, one or both, may suffer; whilst in the female the mammæ, the labia majora, and uterus are the parts occasionally attacked. The tonsils and pharynx may also be involved. When the testicle is diseased, the inflammation involves both the tunica vaginalis and the epididymis; and if the disease be limited to one of these organs, this is situated on the same side with the parotid, solely or most severely, affected. These complications, or metastases as they are termed, usually make their appearance whilst the parotid and the submaxillary glands are enlarged; but, on the other hand, the swellings may decline and disappear from the glands, and not make their reappearance elsewhere until a period, varying from a few hours to one or two days, has elapsed. In this last case, whilst the swellings are in abeyance, active general symptoms, sometimes of an alarming character, may occur; there may be a feeling of great anxiety, pallor of the face, coldness of the extremities, smallness and great frequency of the pulse, and to these symptoms delirium, vomiting, and purging are sometimes added. However, on the reappearance of the local mischief at any part of the body, these symptoms disappear. These metastases, rarely occurring in children, and by no means common in adults, are prone to occur in individuals of the same family; in other words, family idiosyncrasies tend to their development.

The duration of the disease varies; thus, in mild cases it may run its course in four days, whilst in severe cases it may continue for ten days. Its duration is usually longer, and the fever higher, when metastases occur.

PATHOLOGY.—As the disease rarely kills, the opportunities for investigation on this point are necessarily but few. It is generally, however, held that the organ affected, and the cellular tissue within and around it, are inflamed, and that there is an excess of serosity in these parts.

The glands may remain somewhat enlarged and hardened for a considerable time after all the acute symptoms have disappeared, and it is even stated that in rare cases the affected testicle has atrophied.

In Mumps, have we primarily a general disease of which the local effects are the sequence; or, on the other hand, is the disease in the first instance local, and are the general symptoms dependent on such local mischief? In the present state of medical knowledge, this question cannot be answered. Suffice it to say that, whilst some diseases, such as typhoid fever and dysentery, were formerly thought to be primarily "general," further observation on these affections has at least rendered it possible that both of them are in the first instance "local," and that the general symptoms are secondarily dependent on these local lesions. Should this be fully established, it will go far to render probable that most, if not all, diseases are at their commencement local, and, amongst others, the disease under consideration. In favour, however, of the older view, the author may mention the following case in which, after the temperature of the body had become normal (*i.e.* after all fever had disappeared), the right parotid, which had previously remained healthy, began to enlarge, became painful, and corresponded in all respects in its behaviour to the left, this latter gland having been previously affected during the time that the temperature of the body was raised. As no subsequent elevation of the temperature occurred, in this case at least, the local mischief in the right parotid was insufficient to elevate the temperature, and was certainly in point of time sequential to the general condition.

Most common between the ages of five to fifteen, the liability to the disease rapidly diminishes in those under or above these ages; and whilst old age does not afford an entire protection from the disease, it is unknown in children under one year.

It occurs with equal frequency in both sexes. Some authors, however, assert that it is more commonly met with in boys than girls.

It is a contagious disease, not usually recurring a second time, subject to epidemic influence, said to be most common in spring and autumn. Its period of incubation varies from eight to twenty-two days.

Being a disease of short duration and of slight intensity, the patient usually recovers quickly both strength and weight. As in convalescence from all acute affections, so with Mumps, the rapidity of recovery from the anæmia, loss of flesh, &c. is proportionate to the age, being quicker and more perfect in young than old people, and is, moreover, determined by the previous health of the patient: if this has been good, the recovery is accomplished perfectly and with rapidity; whilst, on the other hand, if the health has been impaired by excesses of any kind, or by bad hygienic conditions of life, or if the patient be the subject of chronic disease, or of the tubercular or scrofulous diathesis, the restoration to perfect health is much retarded.

DIAGNOSIS.—This is rarely difficult. An acute febrile disease, accompanied with a swelling in, and assuming the shape of, the parotid gland, is diagnostic of Mumps.

Parotid bubo[1] may in some respects simulate this disease. It is, however, a rare affection; almost invariably follows in the course of one of the acute specific fevers. The swelling does not take on the shape of the parotid gland, and quickly gives evidence of the existence of pus in various parts of the swelling. The mamma, testicle, &c. are never affected.

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[1] My friend Dr. H. Jeaffreson, late Resident Physician to the London Fever Hospital, has supplied me with the following notes concerning parotid bubo. It begins below the ear and behind the jaw, is hard and brawny to the feel, and ill defined. The skin over the swelling is almost invariably of a dusky red colour; is immovable over the swelling; in three or four days becomes boggy, and in five or six bursts and discharges pus. It is by far most common in persons of middle or of old age. It is very rare in children, but may occur at the age of five or six.

Of the acute specific fevers, it almost invariably follows typhus. No case has occurred for some years past at the Fever Hospital after measles, scarlet fever, or typhoid fever. Not more than 3 per cent. resolve. Both parotids may be attacked, and the submaxillary glands may also be attacked in conjunction with, or independently of, enlargement of the parotid. The former condition is by far more frequent than the latter.

Enlarged lymphatic glands situated in the neighbourhood of the parotid perhaps ought to be mentioned, though a careful examination would at once establish the nature of the swelling. Thus there is often more than one enlargement, usually the size of a Barcelona nut, commonly situated immediately in front of the ear. There is no swelling behind the ramus of the jaw; the tumours can be felt to be superficial to the parotid, over which they are mostly movable, and moreover these enlargements of the lymphatic glands are always due to some irritation in the neighbourhood, which can generally be discovered.

Treatment.—In common with the other acute specific fevers, Mumps has hitherto failed to be arrested in its course by any mode of treatment at present known. The utmost therefore that can be done, is to mitigate the severity of the symptoms, and thus conduct the disease to a favourable termination. Being mostly a disease of trifling importance, but little danger is to be apprehended and but little treatment is required.

All active treatment, whether general or local, is in most cases to be avoided. At the very commencement, an emetic followed by a purgative will be generally useful. The bowels should be kept regularly open, but active purgation should be avoided. By this means the severity of the fever is lessened, for it is well known that constipation has the effect of elevating the temperature in febrile patients.

In Mumps, as in all fevers, we have an undue elevation of the temperature of the body, due in part at least to increased combustion of some of the tissues. The treatment must be directed so as to control this increase of waste, and supply the loss by appropriate food. This is especially needful with patients whose health previous to the disease was impaired; for in such, a great amount of bodily and mental weakness, lasting for a considerable time, may result.

With this object in view, two chief points should be attended to; namely, rest and the appetite.

Rest and even confinement in bed should be enjoined; for it is found that in all febrile diseases, exercise, both of mind and body, is capable of increasing the abnormal elevation of the temperature. The pain also, which may accompany febrile disease, is much allayed by perfect rest.

The appetite must have strict regard paid to it; for the increased waste of the tissues is compensated for in proportion to the amount of food digested. Should no food be taken or assimilated, the patient is placed in all respects in the position of a starving person, and to this must be added an active, increased consumption of the tissues.

To secure or promote the appetite and proper digestion of the food, attention must especially be paid to pain, sleep, and the nature of the diet.

Pain, if severe, destroys entirely the appetite and arrests the digestion of food ; therefore, should the pain in the affected organ be great, appropriate treatment must be employed. For the most part, hot fomentations or poultices are sufficient for the purpose. Should, however, these fail, one or two leeches applied in the neighbourhood of the affected organs will generally quickly afford great or even entire relief. This is especially the case when the testicle is affected. The pain is further mitigated by perfect rest of the part; thus the jaws should be moved as little as possible, and the testicle, when it is affected, should be carefully supported.

In regard to the second point—sleep—this can in most cases be attained by easing the pain of the affected part in the manner just described, and by allaying any distressing symptoms that may be present, such as thirst, heat of skin, &c. The thirst can be removed by sucking ice, or the patient may be directed to rinse out the mouth with cold water, or to swallow small draughts of cold water slowly, and at short intervals. It may be much relieved by drinking acid drinks, especially if weak bitter infusions be added. Thus, "whey, or common water acidulated with currant jelly or raspberry vinegar," or a very light infusion of cascarilla acidulated with hydrochloric acid (Graves), will generally succeed. Large draughts of water should be avoided, as they distend the stomach, and give rise to annoying sensations to the patient, and because they retard digestion. Effervescing drinks, moreover, by distending the stomach, are apt to distress the patient. Acid fruits will allay thirst, but they must be used with moderation, as they may produce diarrhœa, flatulence, colic, and even nausea.

The disagreeable sensation due to the hot, dry skin, may also increase the restlessness of the patient and thus prevent sleep; this can be allayed by sponging the body with cold or tepid water, one part of the body only being exposed at one time, to prevent the bad effects of cold. Soap may be added with advantage to the water, as it cleanses the skin more thoroughly and removes any smell that may be connected with the cutaneous excretion; this is advantageous, as it is well known that smells of any kind, and especially when disagreeable, lessen the appetite, and may cause headache, nausea, and even vomiting and much depression. And lastly, strict attention should be paid to the diet, both in regard to its nature and the time and method of its administration. The food should be liquid, so that mastication is unnecessary; thus the inflammation in the parotid is not increased by the movement of the parts, but at the same time it should not be too dilute, otherwise digestion is impaired. Thus, good beef-tea, strong mutton or veal broth, gruel, or arrowroot, milk and eggs, may be given, the latter with caution, as eggs often disagree with febrile patients. Pounded meat, either cooked or raw, and without spices, are also serviceable. Liebig's beef-tea will in some instances be found especially beneficial; this, indeed, can often be tolerated by the stomach when all other foods are rejected.

In respect of time and manner of administration, it must be borne in mind that the digestion of febrile patients is mostly impaired, and therefore only small quantities of food should be given at one time, and these should be taken at the ordinary meal times, and at no others. When prostration is great, and the quantity that can be given at one time is very small, the food must be administered at shorter intervals, and sometimes even every half-hour.

It is inadvisable that the patient should take to allay the thirst any nutritious food, such as milk, as the appetite for food at the proper time is thus much lessened.

During the grave general symptoms that sometimes occur after the disappearance of the swelling in one organ, and before another is involved, more active treatment may be adopted, though mostly the disease passes on to a favourable termination.

If the pulse be weak, the surface cool, and the features nipped, chloric ether, musk, wine, and brandy should be given, and warm baths with mustard to the extremities may prove of service.

SUDAMINA AND MILIARIA.

BY SYDNEY RINGER, M.D.

ALTHOUGH Sudamina and Miliaria generally occur at the same time, they differ so much in respect of their appearance and the method of their development, that they require separate description.

It may be first stated that, while Sudamina often occur without Miliaria, the latter, on the other hand, are generally accompanied by Sudamina. The probable reason of this will afterwards appear.

SUDAMINA.—These vesicles are minute and highly transparent, spherical or oval in shape, and often appear deeply seated in the cuticle. They may sometimes be so small as easily to escape notice, or, on the other hand, they may measure two lines in diameter at their base.

Partly on account of their minuteness, but chiefly owing to their great transparency, they are apt to escape notice. They are best seen when looked at obliquely, and may often be more easily detected by the touch than the sight.

These vesicles are sometimes widely, if not equally, scattered, but at other times grouped and limited to particular portions of the surface of the body. The skin at their base and in their neighbourhood is usually unaltered, whereas, in rare cases, a narrow rim of redness is seen around them. They vary greatly in number; being sometimes so few that they can be easily counted, and at other times so numerous that the chief part of the trunk is covered with them. The base of the neck, the neighbourhood of the navel, and the sides of the thorax, are the parts mostly affected. They reach their full development in a few hours, remain so for about one day, and then either burst or dry up. When of large size and at the height of their development, they look tense and full, and feel hard and "shotty." As they decline they lose their tenseness, and the cuticle covering them becomes wrinkled and loose, at the same time they may extend laterally, and lose their regular form. If they be numerous, they even coalesce, and hence in rare cases rather considerable patches of cuticle may be detached from the corium beneath, the cuticle itself retaining its continuity. On their disappearance, the cuticle forming them is detached from the surface of the body, leaving the skin entire beneath.

If the conditions producing them continue, fresh crops appear, and run their entire course in three or four days.

These vesicles are most commonly limited to the trunk; they may, however, occur on the extremities, but are rarely seen on the face, hands, or feet. Occasionally they become slightly turbid, but they mostly remain transparent throughout. Their contents, watery, colourless, and transparent, are generally acid, occasionally neutral, and very rarely alkaline; they contain chlorides. No organic elements are seen on microscopic examination, with the exception of a few epithelium cells, probably derived from their cuticular covering.

They are characteristic of no particular disease, but are produced by sweating;[1] and hence they often occur on the decline of fevers, and especially on those "critical days" when the sweating is most profuse.

Much difference of opinion exists regarding the anatomy and the method of production of these vesicles, or Sudamina. According to some authorities they are due to accumulation of sweat in obstructed and distended sweat ducts. Others, Bærensprung for instance, hold that they are produced by the exudation of the perspiratory fluid between two layers of the cuticle, the exudation being caused by obstruction of the ducts from accumulation of effete epithelium cells.

As Sudamina produce no annoyance or symptoms, no treatment is required. They are best prevented by checking as far as possible the sweating that produces them. This sweating, as has been stated, is often produced or increased by weakness and impairment of the health, and may be reduced by relaxation from work, outdoor exercise, sea-bathing, and tonics.

Dr. Druitt states that the sweating of hectic fever can be controlled for some hours by the sponging of the body with water as hot as can be borne. In many cases of phthisis all treatment fails to lessen the amount of perspiration, for in this disease the perspiration is caused both by the daily fall of the temperature and the exhaustion produced by the disease.

MILIARIA.—The vesicles of Miliaria, in both their appearance and method of development, differ from those of Sudamina. They are like the latter, however, in size, and are produced by perspiration.

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[1] Sweating is most commonly due either to a fall in the temperature of febrile patients or to general weakness and exhaustion. It especially occurs towards the close of typhoid fever, during the early convalescence of scarlet fever, and in the course of phthisis, this last-named disease being often accompanied by great daily variations in the temperature. In scarlet fever, according to the author's experience, profuse sweating is more common, and lasts longer during the early convalescence of the patient than in other febrile diseases; and hence Sudamina, in very large quantities, are often met with at that period of the malady. Profuse sweating at the commencement of febrile diseases (with the exception of rheumatic fever), when the temperature remains permanently high, indicates great weakness, and thus adds to the seriousness of the prognosis.

In non-febrile persons, if sweating be easily produced by excitement, exertion, or sleep, the health is generally impaired. It must, however, be recollected that great differences in respect of the amount of sweating are met with in different individuals. Persons who have returned from tropical climates often continue to sweat greatly on the slightest provocation.

They are at first acuminate in form, and round or oval at their base; but in the course of twenty-four hours they sometimes extend and become irregular in outline. They never present the tense rotund appearance of Sudamina. They are almost invariably surrounded at their base by a narrow rim of redness, and the surface on which they are seated is not infrequently somewhat elevated. Their contents are from the first turbid, opaque, and white; are acid in reaction, and by means of the microscope are seen to contain a large number of granular cells, often shrunken-looking, as if badly developed. On the addition of acetic acid, the granules disappear, and there are seen one, two, or three nuclei. The vesicles of Miliaria, when freely exposed, quickly dry up, and a little redness remains for a short time longer. The skin between these vesicles is often mottled with redness, and here and there small red papillæ are seen, on the summit of which, by means of a lens, a small vesicle can often be detected.

Vesicles, in all respects similar to those last described, are not infrequently seen during the eruptive stage of scarlet fever, and they are situated on the papillæ of the skin, which are elevated in this disease. They are formed only when the rash is intense, and on those parts most affected; and are therefore seen most frequently under the clavicles and around the navel. They may, however, occur on the extremities, and are then best developed on the thighs. They are often arranged in elongated groups, corresponding to the furrows of the skin. Miliary vesicles are often abundant on the surface of patients who suffer from rheumatic fever, when sweating is profuse, and especially when this has an offensive smell. Hence, in this disease, they are most developed and abundant in young adults, and are rarer in children and old people. They are probably produced by inflammation. This is shown by the large quantity of cells they contain.

To the author it appears probable that the vesicles of Miliaria are not formed during the act of sweating, but that they result from the irritation which the sweat causes. This view is supported by the following considerations:—The vesicles are especially apt to occur on those parts of the body from which the free evaporation of the sweat is prevented. Thus they are found under the band of the drawers when seen nowhere else; and in cases of profuse sweating, if a piece of flannel be worn for some time, firmly tied round the neck (at which part of the body these vesicles are easily produced), they not infrequently appear—ceasing, however, to be formed when the flannel is removed. Moreover, in rheumatic patients these vesicles are very abundant over the back, at which part the perspiration is confined, and often allowed to accumulate, because of the pain caused in such movement of the patient as would be required for washing this part of the body. They are also most numerous in those rheumatic patients whose sweat is usually offensive and disagreeable. And further, according to the author's experience, they are most apt to occur in other patients when, from impairment of the health, either by overwork, want of sleep, excess of smoking, or other causes, the sweat smells offensively, and when probably it causes greater irritation of the skin.

For the further settlement of this question the following experiments were made :—Dry spongio-piline was placed on various parts of the body, especially round the neck, and kept on the surface several days. By the action of this dry spongio-piline, Sudamina, but in no case Miliaria, were produced. On the same patient, and to the same parts, linseed-meal poultices (which contain much acrid resin) were applied, and vesicles of Miliaria were often produced, and especially on the neck. Bread poultices, which are less irritating, produced these vesicles, but in a much smaller number. In favour of this view may be further advanced the fact, that the vesicles of Miliaria are often accompanied by troublesome itching. This is most marked over the back, the part most dependent in rheumatic patients, and it may be very annoying, and prevent sleep.

In the treatment of Miliaria but little is required. Frequent sponging of the surface of the body with soap and tepid water lessens their production, and removes the itching.

VARICELLA.

By Samuel Jones Gee, M.D.

Definition.—A contagious, febrile disease which is attended with an eruption of vesicles, does not last longer than a week, and does not recur in the same individual.

Just as small-pox and measles were not at first distinguished from each other, and mediæval measles included scarlet fever, so were small-pox and chicken-pox confused together until the last century. In the year 1730, appeared what seems to be the earliest assertion of the doctrine of non-identity; we read that "the pestilence can never breed the small-pox, nor the small-pox the measles, nor they the crystals or chicken-pox, any more than a hen can breed a duck, a wolf a sheep, or a thistle figs, and consequently one sort cannot be preservative against any other sort." So far, Fuller. In the well-known paper read before the College of Physicians in 1767, Heberden enumerates what were to him sufficient reasons for disallying the two diseases. Yet since that day there have never been wanting those who have disputed the validity of the distinction drawn; indeed the doctrine of Fuller and Heberden has hardly taken fair root in any country except their own.[1]

Let us review the arguments in favour of the non-identity of chicken-pox and small-pox.

1. Chicken-pox and small-pox are not interchangeable:—

(i.) By infection. (A) There is not a single authentic instance on record wherein either of the diseases was the result of exposure to the infection of the other. (Trousseau.) (B) Chicken-pox may prevail as an epidemic isolated completely from cases of small-pox. (Möhl.) Now, an epidemic of varioloid, free from concurrent examples of non-modified small-pox, has never yet been seen.

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[1] In the first edition of this volume there stood in this place a detailed criticism of the arguments, in favour of the identity of chicken-pox and small-pox, adduced by Hebra, the living Coryphæus of that doctrine. Since Hebra's book has been translated by Dr. Fagge for the New Sydenham Society, the reader who wishes to know what Hebra has to say may easily procure that information. I confess that I was annoyed at one or two of his assumptions, and amused at his logic; I marvelled that a man with so great a reputation should write so loosely. Was Heberden a physician without experience, Gregory a mere apprentice to his art, and Trousseau's opinion unworthy of consideration? If Hebra will confuse varicella and varioloid, he must even do so; however, let it not be said that he does not deny the existence of varicella as a distinct disease. But 'quandoque bonus dormitat Homerus' shall cover all faults.

(ii.) By inoculation. (A) Chicken-pox is not inoculable. (Bryce, Trousseau.) (B) Small-pox, whether modified or not, inoculated, has never yet been proved to beget chicken-pox.

2. Chicken-pox and small-pox are not mutually prophylactic:—

(i.) Small-pox did not prevent the occurrence of chicken-pox. (Heberden: Halford, quoted by Gregory.) The reader need not be reminded that undoubted small-pox very rarely recurs.

(ii.) Chicken-pox does not prevent the occurrence of small-pox. In the Children's Hospital a girl sickened with chicken-pox on January 17, and communicated it to the child in the next bed; in April, the girl first spoken of was attacked with modified small-pox (there were good vaccination marks on her arm), attended by severe invasion symptoms (vomiting, headache, backache), which preceded the eruption several days; she recovered; soon afterwards her mother and sister were laid up with small-pox.

3. Chicken-pox and cow-pox are not mutually prophylactic.

(i.) Cow-pox does not prevent the occurrence of chicken-pox; this we see every day.

(ii.) Chicken-pox does not prevent the occurrence of cow-pox. (Abercrombie, Bryce.)

If the very existence of chicken-pox admits of dispute, we cannot be surprised when we find that the published descriptions of the disease present differences and discrepancies without end. No doubt all physicians who have written upon small-pox, from the days of Rhazes downward, have left some notices of chicken-pox; but these are, with a few exceptions, either vague or confused: vague, because up to the beginning of the last century the two diseases in question were regarded as essentially identical; confused, because hardly had a clear separation been made, before the introduction of vaccination, or rather the exaggerated expectations to which vaccination gave rise, led men to thrust into the realm of Varicella every example of varioloid. And even at the present day, the evanescence of the disease and its lack of all gravity militate against better knowledge; the inducements and the opportunities for study are small indeed.

The epithet *chicken*-pox is derived from *cicer* (chick-pease) through the French *chiche*. Varicella (*varicula*) is a legitimate diminutive of *vărus* a pimple.

CAUSES.—The efficient cause of Varicella is "contagion." The disease is readily communicated through the air to a distance of several yards at least; in degree of contagiousness chicken-pox "seems as infectious as small-pox." (Heberden.)

Chicken-pox has never yet been transmitted by inoculation. Heberden (naturally enough) presumed that the disease was inoculable; the only inference he drew from his failure to inoculate a person who had previously suffered from the disease was that it did not recur. About the end of the last century, the prevailing opinion in France was that chicken-pox could not be so transmitted. The instances of supposed inoculation narrated by Willan are most unsatisfactory; the notion that such transmission might be impossible seems hardly to have crossed his mind. Berard and De Lavit effectively inoculated Varicella, but Varicella which presented the same symptoms, progress, and form of pock, as variola. Heim (quoted by Cross) was not less successful; he took his lymph from umbilicated vesicles which equalled in duration the pustules of small-pox. Next came Bryce, who in 1818 published the result of his attempts to inoculate thirteen persons with the fluid of what, from his description, we may freely admit to have been undoubted Varicella; the operation-wound healed up, and that was all. Lastly, Trousseau has failed in all his inoculation trials.

Chicken-pox is not known to recur. It is a disease of childhood. The following table has been drawn up for me from the records of the Children's Hospital:—

	Under 1 month.	Under 2 months.	Under 3 months.	Under 6 months.	Under 12 months.	Under 18 months.	Under 2 Years.	Under 3 Years.	Under 4 Years.	Under 5 Years.	Under 6 Years.	Under 7 Years.	Under 8 Years.	Under 9 Years.	Under 10 Years.	Under 12 Years.	Total.
Boys	2	2	4	29	45	34	36	36	47	44	33	19	10	4	3	1	349
Girls	0	6	9	28	52	28	39	42	53	52	25	11	19	6	2	6	378
	2	8	13	57	97	62	75	78	100	96	58	30	29	10	5	7	727

Judging from the same reports, there seems to have been an epidemic of chicken-pox in 1856.

Adult females are occasionally attacked. (Gregory.)

DESCRIPTION OF THE DISEASE.—1. *Pre-eruptive Period.*—(i.) *Duration.*—It does not exceed four days, and is certainly less than a week (Gregory): it lasts eight or nine days? (Heberden): from fifteen to seventeen days (Trousseau): my own observations would lead me to place the duration at about a fortnight. (ii.) *Symptoms.*—There are no symptoms to be noticed before the eruption (Gregory): they are absent or slight (Heberden, Möhl): poorliness, headache, and feverishness precede the eruption by a few hours (Trousseau): cough is sometimes observed.

2. *Eruptive Period.*—The eruption appears within the first twenty-four hours of poorliness in the form of small rose spots, slightly acuminated; from ten to fifteen come out on the first day; they appear on any part of the body. (Trousseau.) Heberden and J. P. Frank also describe a red spot as the first appearance of the eruption of chicken-pox. According to Bryce and Gregory, the first thing seen is an eruption of vesicles. For my own part, I have always noticed the vesicle to be preceded by a red spot, and such a spot as

I should not hesitate to call a papule, but a papule due to mere hyperæmia of the cutis vera, and not to an exudation into it, for tension of the skin causes the varicellous papule to disappear.

On the second day there may be a hundred or a hundred and fifty fresh spots; those of the previous day have the epidermis raised in the form of a bleb, sometimes perfectly round, containing serosity as clear as water; there is no inflammatory areola. (Trousseau.) The vesicles from the first have the size of split pease (Bryce and Gregory); at any rate, that size is soon attained or exceeded. The patient has the appearance of having been subjected to a shower of scalding water. (Bryce.) The vesicle is unicellular, not umbilicated, has a very delicate cuticle, and when pricked collapses perfectly; "after the vesicle is emptied, the finger passed over it does not detect any swelling of the cutis vera or the parts beneath." (Bryce, Möhl, Cross, Gregory.) The latter statement seems to me to be much too exclusive; a distinct elevation may often be detected, but is, like the swelling of the rose spot of typhoid fever, dispersible by pressure. The eruption occupies all parts of the body, the hairy scalp not excepted. The shape of the vesicles on the trunk is often oval, the long axis being athwart that of the body. Itchiness is common, and impels the children to rupture the vesicles.

The next morning a hundred or a hundred and fifty new spots will have appeared during the night, the eruption of the preceding day having become vesicular. (Trousseau.) The contents of vesicles which have lasted twenty-four hours become slightly milky; the turbidity, however, is uniform. A slight inflammatory areola appears.

This nocturnal outburst of spots (which become vesicular within ten hours) is repeated for four or five succeeding nights from the beginning of the disease. (Trousseau.)

Many vesicles, as soon as they have attained their full size, get broken, and so encrust at once. Those that remain unbroken present, on the third, fourth, or fifth day of their existence, a small central scab, which quickly attains to the size of the vesicle, and falls in a day or two.

This scab is thin and granular; it falls in fragments, and leaves no enduring redness and no cicatrix. If the vesicle have been subjected to unwonted irritation, the scab may be thick, coherent, and may leave, when it falls, a permanent pit. Gregory never saw a pit left.

3. *Concurrent Symptoms.*—These are of no importance; the tongue is clean, the pulse unaffected, there is no appearance of feverishness. (Heberden, Gregory.) There are outbursts of fever, sometimes violent, for four or five nights, ceasing by day. (Trousseau.) Catarrh is common; it may occasionally be serious, as in the case of a child who was under the care of Dr. West, on account of a chronic swelling of the glottidean mucous membrane; her respiration was therefore somewhat laboured at best; by an attack of chicken-pox she was brought to such straits that for forty-eight hours it seemed as if she could hardly escape tracheotomy; with the cessation of the eruption the urgent dyspnœa ceased.

It is with reluctance that no reference has been made to the name of Willan; yet I think that the reader who consults his book, will find it difficult to believe that at least some of the cases which afforded materials for his descriptions were not cases of small-pox, especially those which were admissible into the variety of Varicella coniformis.

4. *Sequelæ.*—An attack of chicken-pox sometimes leaves children in a poor state of health, such as may not be overlooked.

DIAGNOSIS.—Modified small-pox constantly resembles chicken-pox in (i.) the mildness of the symptoms, premonitory and concomitant; (ii.) the scarcity of the eruption, and its character of coming out in successive crops; (iii.) the shortness of the duration of the disease. (Cross.)

Two or three days' high fever, with vomiting, headache, and light-headedness, before the eruption, would exclude chicken-pox. The absence of those symptoms would not exclude small-pox.

Although the papule of chicken-pox has a certain elevation, it is something unmistakeably different from the peculiar shotty hardness of the papule of small-pox, modified or not. "All cases in which any of the pocks are observed to be indented on the surface, whilst their contents are clear, and before incrustation has commenced, are at once to be distinguished from the water-pox." (Cross.) The base of the varioloid vesicle is hard and raised to a degree never observed in chicken-pox. The pocks of varioloid are not necessarily indented; when not, we must trust to the "greater firmness and less rapid growth, although of equally short duration." "An elevation left after the scab separates determines the question." (Cross.) The commencing scab of Varicella may be mistaken for an umbilication. A perfect vaccination scar (it may not be unnecessary to add) often coincides with varioloid.

The characters assigned by Gregory to his "variola varicelloides," are: that it has at least forty-eight hours of premonitory fever; that there are tubercular elevations of the skin; that an umbilication is always present; and that the scabs differ. The last character is comparatively unimportant, the first and third are put too absolutely.

PROGNOSIS.—"No physician has ever seen a child who has died of chicken-pox; fatal complications are quite independent of the exanthematous fever." (Trousseau.)

TREATMENT.—"Curatur hic morbus quiete animi et corporis, et abstinentia a carne, vinoque." As much as possible, children should be prevented from picking the vesicles and scabs present on the face. Small doses of quinine will be useful during convalescence.

VARIETIES AND SYNONYMS.—(1) Varicella lentiformis. (Willan.) On the first day of the eruption appear flat red elevations, in the centre of each of which a vesicle is soon formed; the vesicle never exceeds the tenth of an inch in diameter; the scab falls without leaving a scar.

(2) Varicella coniformis (Willan); swine-pox. The vesicles rise suddenly, and have a hard inflamed border; on the second day the surrounding inflammation is more extensive; on the third the fluid is purulent; a permanent scar results from each pock.

(3) Varicella globularis (Willan); hives. The vesicles are larger than in varic. lentif. and the cutaneous hardness less than in varic. conif.

(4) Varicella sine varicellis. (Wilson.)

(5) Varicella solidescens, verrucosa, papularis, variola cornea (Van Swieten), pemphigus variolodes solidescens (J. P. Frank), stone-pox, horn-pox, or wart-pox, is a form of true small-pox.

(6) Varicella cellulosa (Cross), pustular umbilicated varicella (Wilson), variola varicelloides (Gregory), are names for that form of modified small-pox which most resembles Varicella.

Synonyma of true Chicken-pox.—Crystalli (Ingrassias); variolæ crystallinæ, spuriæ, volaticæ, benignæ (Morton); lymphaticæ (Sauvages), pusillæ (Heberden); pemphigus variolodes vesiculosus (Frank); varicella bullosa (Cross); water-pox, water-jags (provincial, to dag, daggle = to sprinkle).

The following are some of the best accounts of the disease:—

Wm. Heberden: 1st. Med. Trans. Coll. Phys. vol. i. 1768. 2d. Commentarii, 1802.

Jno. Cross: Hist of Variolous Epidemic in Norwich, 1820.

Nicol C. Möhl: De Varioloidibus et Varicellis, 1827.

Geo. Gregory: 1st. Cyclop. Pract. Med. vol. iv. 2d. Lectures on Eruptive Fevers, 1843.

A. Trousseau: Clinique Médicale, vol. i. 1861.

SMALL-POX.

By J. F. Marson.

Definition.—Small-pox is a febrile, eruptive, and infectious disease, the product of a morbid poison; which, after a period of latency, causes the development of an eruption on the surface of the body; this passes through the stages of pimple, vesicle, pustule, and scab; and, as a rule, exhausts or destroys the susceptibility to the disease, in the same person, for the remainder of life.

Synonyms.—Jadarí, Arabic; Εὐλογία, Modern Greek; Variola, Latin; Small-pox, English; the Pocks, Scotch; Galra breac, Irish; Petite Vérole, French; Blattern, German; Vaiuolo, Italian; Viruelas, Spanish.

History.—The origin of Small-pox is involved in much obscurity. A great deal of labour and learning have been bestowed in endeavouring to trace the beginning of the disease, but seemingly without any very decided success. The ancient Greeks and Romans do not appear by their writings to have been acquainted with Small-pox, although De Haen, Dr. Willan, Mr. Moore, and Dr. Baron, have endeavoured to prove the contrary. Dr. Friend, Dr. Mead, and Dr. Mason Good were of opinion that the disease was not known to the ancient Greeks; and Dr. Adams, in the Appendix to the Commentary on Book Second of his translation of Paulus Ægineta, agrees entirely with the opinion of Friend and Mead, that the disease was not known to the ancient Greeks.

One of the earliest notices of a disease exhibiting the striking characteristics of Small-pox is to be found in the historical writings of Procopius,[1] who lived in the middle of the sixth century. The disease[2] "began A.D. 544, at Pelusium in Egypt, from whence it spread to Constantinople. This corresponds closely with the era commonly assigned in medical books to the first appearance of Small-pox, viz. A.D. 569, the year of the birth of Mahomet. In that year an Abyssinian army, under Abrahah the viceroy, appeared before Mecca, and was unexpectedly compelled to raise the siege. Several circumstances concur to render it probable that the sudden retreat of the army was owing to the breaking out of Small-pox, and the dreadful mortality which it occasioned. Bruce,[1] in his travels, met with a manuscript account of the war, which confirms this story, and strengthens the opinion that Small-pox first appeared in Egypt and Arabia about the middle of the sixth century."

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[1] De Bello Gothico, lib. ii.

[2] Gregory, Cyclop. of Pract. Med. vol. iii. p. 735.

Paulus Ægineta lived at the end of the sixth or beginning of the seventh century. He professed to have treated, in his seven books, on all subjects connected with medicine and surgery, yet he says not one word to lead us to believe he was acquainted with Small-pox.

Rhazes, an Arabian physician, who flourished about 910, is generally referred to as one of the earliest and best writers on Small-pox. No doubt the disease had existed for some time before he undertook to describe it; indeed, there is evidence in his work that it had, and he alludes to others who had written about it, especially to Ahron of Alexandria, and Messue of Bagdad.

In the East, whence we receive the earliest accounts of Small-pox, there is a tradition—a mere tradition[2]—that the disease in man had its origin from the camel. It is well known that this patient animal is extensively used in Arabia, Egypt, &c. as a beast of burden. If we may venture to reason on the subject from analogy, the tradition is not likely to be correct, inasmuch as all other diseases that have been conveyed to man from the lower animals are not communicable by infection, only by inoculation. When once produced in man, they are still not infectious, in the usual acceptation of the term, as Small-pox is; only producible again, from one to another, by inoculation, as in the instances of cow-pox, glanders, hydrophobia, &c. When Small-pox appeared in this country in the sheep in 1847,[3] we tried to communicate it, by inoculation, to the human subject, and thought we had succeeded in doing so, and the virus was carried on from one to another for several weeks in succession. The pock produced was very like cow-pox, having only, as we thought, a bluer tinge, and was protective against Small-pox, as we ascertained by inoculating the patient afterwards with the lymph of human variola: but we had unfortunately used for the original *ovination* the same lancet, instead of having a new one, as we ought to have had, that we had previously used for vaccinating; and although it was, as we believe, perfectly clean, and free from vaccine lymph, nevertheless, as the disease could not be produced again in the human subject, either by Mr. Ceely, of Aylesbury, who made repeated trials with the lymph of sheep-pox, or by ourselves, the experiment was never brought before the medical profession. Sacco writes of having frequently succeeded, in Lombardy, in transferring the virus of sheep-pox to man, and that it was as successful in protecting against Small-pox as cow-pox is. There has never been any reason to suppose that the Small-pox in sheep has produced by infection any disease in man.

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[1] Travels to discover the Source of the Nile, vol. i. p. 514.

[2] Moore's History of Small-pox.

[3] For an interesting and able account of Variola Ovina, or Small-pox in Sheep, see a work by Professor Simonds, of the Royal Veterinary College, London, 1848.

Those readers who are desirous of further information on the early history of Small-pox, will find a great deal of interesting reading on the subject in Moore's History of Small-pox; Willan's Inquiry into the Antiquity of the Small-pox, Measles, and Scarlet Fever; Baron's Life of Jenner; Monro's Observations on the different kinds of Small-pox; and in Dr. Greenhill's translation of Rhazes, forming one of the volumes of the Sydenham Society's publications.

Description of Small-pox.—The disease is divisible into varieties, which, for convenience, may be described separately.

1. *Variola Discreta;* 2. *Variola Semiconfluens;* 3. *Variola Confluens;* 4. *Variola Corymbosa;* 5. *Variola Maligna;* 6. *Variola Benigna;* 7. *Variolæ Anomalæ,* or irregular forms of the disease, embracing those instances in which Small-pox is complicated with other diseases.

It is called *discrete,* when the pustules stand separately; *semiconfluent,* when they partially coalesce; *confluent,* when they join and run into each other; *corymbose,* when the disease appears in patches; *malignant,* when the eruption, besides being, generally, confluent, the initiatory and succeeding symptoms are very severe, with hæmorrhage from the mucous surfaces, patches of purpura, and discolorations of the skin as if from having been bruised; *benign,* when, although perhaps confluent, the eruption is superficial, and the accompanying symptoms are of a mild character; *anomalous,* when the disease is complicated with other diseases, eruptive or otherwise, as measles, scarlatina, urticaria, &c.—or pneumonia, hooping-cough, bronchitis, disease of the brain, mania, &c.

Small-pox is divisible into four stages:—1. The stage of incubation, which lasts twelve days, from the date of receiving the variolous germ. 2. The stage of initiatory or eruptive fever and invasion, lasting forty-eight hours. 3. The stage of maturation, continuing about nine days. 4. The stage of secondary fever, desiccation, and decline, lasting, of course, an uncertain time; varying according to the severity of the disease.

1. *Stage of Incubation.*—Small-pox appears on the skin on the fourteenth day after the infection of the disease has been received into the constitution, the precise time being after thirteen times twenty-four hours have elapsed from the moment of taking the disease; this time will of course occupy twelve whole days, and part of two others. It is believed by the writer that the time from taking the disease to its appearance on the skin is never longer than fourteen days, and his attention has been constantly directed to the subject for upwards of twenty years. It is true but very few cases afford a decided opportunity for judging of the precise time of incubation, not above one perhaps in fifty or more, but still these few cases are the very cases of value in deciding the point; they have to be watched for carefully to

be found. Three or four instances have occurred in which it seemed likely the disease had appeared between the tenth and eleventh days after receiving the infection; they were cases occurring after vaccination, under which condition other stages of the disease are often interrupted, or cut short; still these cases, seemingly decided cases, have been so few they can hardly be relied on: but the others, on the contrary, so many in the aggregate, they can hardly have failed to indicate the true time.

2. *Stage of Primary Fever.*—The ordinary course is this,—after twelve days' freedom from illness, there is severe indisposition for forty-eight hours, and then the eruption of small-pox begins to appear. This is almost the invariable course. Still it is not invariable. In a few cases, but very few, there is more or less illness all through the period of incubation. The patient has not been so well as usual: experienced even at the time of taking the disease some unpleasant sensation, felt some nausea, or giddiness, or sense of alarm, without knowing why it had happened.

3. *Stage of Maturation.*—In distinct and semiconfluent Small-pox the early constitutional symptoms are very much ameliorated on the third day, or about that time, when the eruption has been developed on the skin; and the same remark applies with some reserve to confluent cases, but not so completely as to the distinct and semiconfluent forms of the disease; the development of the eruption affords only partial relief in confluent cases. The eruption appears first, usually, on the face, forehead, and wrists, and then on the rest of the body; it is generally a couple of days later on the legs and feet than elsewhere. It is not thrown out at random, without order; it may be observed to be in threes and fives, forming crescents, and in some instances, when it happens that two crescents come together, they form a complete circle. The eruption is at first papular, then vesicular, then pustular, and takes about eight days to arrive at its full development, before the pustules begin to discharge their contents. During the stage of maturation, or concoction as the older authors termed it, there is often considerable swelling of the face and eyelids, so that the patient is popularly said to be blind with Small-pox for a certain time: and there is ptyalism in many cases, and in some a very tender state of the skin, so tender that the patient complains of the pain from the act of merely feeling the pulse; all these may be looked upon as favourable signs of the disease; patients who have the face a good deal swelled for four days, who have pretty free salivation, and a very tender skin, nearly always do well.

4. *Stage of Secondary Fever, Desiccation, and Decline.*—When Small-pox is not of such severity as to destroy life by the eighth or ninth day of eruption, there is a great increase of fever again, called the secondary fever, which is of vast importance, and gives rise to a train of severe and complicated symptoms, which will be described under the heading of *Secondary Fever.* Concurrently with it the pustules discharge their contents, and form dry, scaly scabs, and

in favourable cases the disease begins to decline; especially in those cases which will be described under the term *Variola Benigna*, and in modified Small-pox, as it is now frequently seen after vaccination.

VARIETIES OF SMALL-POX.—1. *Variola Discreta*, or *distinct Small-pox*, is a term applied to that form of the disease in which the pustules stand separately, or apart from each other, and might be readily counted. It is the simplest form of the disease, and is hardly ever attended with danger to life, except in children, who may be cutting teeth at the time, and may have convulsions or some affection of the brain produced, it may be, by the combined influence of Small-pox and teething.

2. *Variola Semiconfluens* is that form of the disease in which the pustules partially coalesce, cannot be said to be distinct from each other, nor yet to run generally into each other. It is readily distinguished in practice. Patients with this form of the disease usually do well; when it proves fatal, the cause is from the combination of circumstances above alluded to in children with the distinct form of the disease; or else from some complication, as erysipelas, gangrene, &c., or, as happens now and then, from the petechial or malignant form of the disease being associated with only a semiconfluent form of eruption. The amount of eruption does not alone destroy life in semiconfluent as in confluent Small-pox.

3. *Variola Confluens.*—This is the form of the disease which destroys the greatest number of persons; the danger in fact arising principally from the amount of pustulation. It is found to prove fatal at the Small-pox Hospital, when large numbers are taken into account, at the rate of 50 per cent. From the first the papulæ are very numerous, countless, and as the disease advances the pustules run into each other, and in the worst cases form one mass of disease. Even the confluent form of the disease may be fairly said to have its varieties. When the disease is but just confluent, and the patient has been previously in good health, with an unimpaired constitution, he will probably recover. Patients do so in fact, as previously stated, at the rate of 50 per cent.; but when the disease is severely confluent, when it is almost impossible to put the end of a pencil between the pustules in many parts of the body, especially on the face,—and such cases are often met with,—a quarter of the amount of pustulation would be enough to destroy life. In cases where the eruption is observed at first to be generally confluent from head to foot, there may be said to be but very little chance for the patient's recovery. The danger is always rendered greater, *cæteris paribus*, when the eruption is very full about the head, face, and neck.

The marked difference, from the first onset of the disease, between the distinct and confluent varieties of Small-pox, cannot fail to strike all observers. In the confluent form the initiatory fever is more intense, there is often delirium, sometimes of a very violent and uncontrollable kind, especially in persons accustomed to live freely, and in those more especially in the habit of indulging in taking ardent spirits. Such persons often require to be put under restraint to prevent their injuring themselves or others. They are impressed often with the belief that they are about to be murdered, and endeavour, accordingly, to escape from control,—and have a tendency to commit suicide; therefore it is desirable to put knives, razors, &c. out of their way. The nervous system is implicated; there are tremors of the hands and lips—a state, in fact, often bordering on *delirium tremens*, produced partly by the Small-pox, and partly by the previous habits of living. Draymen, barmen, potmen, tailors, and the women on the town, are very unfavourable subjects to be attacked with Small-pox, owing to their habits of indulging freely, and almost daily, in strong drinks. A very large proportion of the patients die who suffer in the early stage of confluent Small-pox from delirium; it should be looked upon in every instance in which it occurs as a very unfavourable symptom.

Fortunately, all cases of confluent Small-pox are not of this dangerous kind. There is the confluent superficial eruption, which often goes through its course without an untoward symptom, especially in persons lately from the country, whose health is unimpaired by the injurious habits and bad air of a town life.

Sometimes the pustules in confluent cases are very large and flat, they do not accumulate well, the edge of them is not well defined, and after they have been out some days they have a tendency to spread out, to become larger. These are dangerous cases, and usually end in death.

On the top of each pustule, or on many of them, a dark spot is formed during the stage of maturation in some cases of confluent Small-pox, and it will be observed, when this occurs, the pustules do not acuminate well, they are rather flat; whenever these signs are noticed, the case should be looked upon as one of great danger, and the patient will most likely die.

Persons of a weakly constitution, those especially with fair hair, have sometimes, about the eighth day of eruption, large bullæ, filled with serum, intermixed with the small-pox eruption. This is an unfavourable sign; such patients require wine, beef-tea, jellies, &c., early, almost as soon as this symptom is observed.

In some cases of confluent Small-pox there is an absence of the damask rose-red areola described, and very correctly, by the old authors, as surrounding, for a short distance, each pustule of the disease; and, instead of this, the skin between the pustules is generally inflamed from head to foot. These cases always do badly.

The watery-pock is another dangerous variety of confluent Small-pox. These are, usually, very offensive cases: are accompanied with a good deal of secondary fever; and end fatally for the most part, or else there is a very tedious convalescence.

The eruption of Small-pox is formed on some of the mucous surfaces as well as on the skin generally,—in the mouth, on the tongue, in the nares and fauces, on the membrane lining the larynx, trachea, and bronchi. When the vari are numerous on the larynx and trachea, the danger of the patient is thereby very much increased; they produce a viscid secretion, cough, and a peculiar hoarse, metallic sound in coughing, indicative of their presence in these parts. A constant subject of complaint in most cases of confluent Small-pox is the soreness of the throat. To nearly every patient it is necessary to explain that this inconvenience is caused by the eruption being formed on the roof of the mouth, soft palate, in the fauces, &c., and that it is impossible to interrupt its course there any more than on the surface of the skin generally. Still the inconvenience goes off considerably in a few days; the vari on the mucous surfaces have a shorter duration than on the skin generally; do not maturate and scab as on the outer skin, from being constantly kept moist by the natural secretion of the mucous surfaces; they never reach beyond the stage of vesicles.

4. *Variola Corymbosa.*—This is a very singular and very fatal form of the disease. It is rather rare. It is called corymbose, from *corymbus*, a bunch or cluster of ivy-berries, &c. *Corymbose* is also a botanical term, applied to a class of plants, the flowers of which are formed in clusters, like those of the carrot. We have gone over the register of the Small-pox Hospital for thirty years, for the purpose of investigating minutely the danger to life in this form of the disease, and find that, in this time, 104 cases of corymbose Small-pox have been admitted—29 in unvaccinated persons, 74 in the vaccinated, and one after inoculation. Of the 29 unvaccinated persons, 13 died, or 44 per cent.; of the 74 vaccinated, 32 died; and, deducting 2 who died of superadded disease, there remains a mortality of 41 per cent. The single case of corymbose Small-pox after inoculation died. It will thus be seen that corymbose Small-pox is, in all cases, a very fatal form of the disease, and brings life into danger nearly as much in vaccinated as in unvaccinated persons, varying only about 3 per cent. The danger in this form of Small-pox often seems to be out of all proportion to the amount of pustulation, which rules so powerfully in other forms of the disease: why it is so, it is impossible with our present knowledge to say, and it is probably one of those things which will for ever remain inexplicable. The disease, as stated, appears in clusters, or, it may be, that only a single cluster is formed, and yet the fatal character before alluded to is given to the disease. In other parts of the body the eruption is perhaps but sparsely scattered, and we might expect the disease to rank in danger with a common semiconfluent case; such, however, is not the fact in practice. It generally happens there are two or three patches, about the size of the palm of the hand, in different parts of the body, in which the pimples are as closely set as could be; and in the immediate neighbourhood of each patch, the skin is for some distance free from eruption, or nearly so, a few spots only of the disease being formed. There is a great tendency to symmetry in this form of the complaint; when a patch is formed on one arm, or leg, it often happens that a similar patch is formed on the same part of the corresponding limb on the opposite

side. In some instances there are numerous corymbose patches in different parts of the body, about the size of a half-crown or five-shilling piece. When these corymbose cases seem to be recovering, very frequently some dangerous complication arises to mar our fair hopes of a successful termination of the malady, and generally, under more favourable circumstances, there is a long and tedious convalescence.

5. *Variola Maligna.*—This truly frightful variety of Small-pox was called by the early writers on the disease Black Pock, or *Variolæ Nigræ.* The symptoms are very formidable at the onset. The blood appears to be poisoned from the first by the disease; it is rendered very fluid and watery. If a portion be drawn from a vein, a large part of it will be found to be serum, and what ought to be crassamentum remains almost fluid; it is principally colouring matter,—the fibrin seems to have disappeared. The countenance of the patient is sunken, the breathing anxious, and in some instances death takes place before the eruption has been developed, leaving some doubt about the real character of the disease in the minds of those persons not by practice familiarly acquainted with its varied appearances;—a doubt whether it was Small-pox, scarlet fever, or some other form of idiopathic malignant fever.

The eruption in malignant Small-pox is rather slowly developed. There is hæmorrhage from some, occasionally from all, or nearly all, of the mucous surfaces; from the nose, from the mouth, from the air-passages, from the bowels; the urine is high-coloured from blood mixed with it. In the female there is invariably hæmorrhage from the uterus, and abortion in cases of pregnancy. The fœtus is usually born dead. Early in the attack there is a patch of effused blood under the conjunctiva, which should always be looked upon as a most dangerous symptom. We have seen blood, in some very rare instances, ooze from the ears and eyes. Livid patches from effused blood are formed on the surface of the body, and blood is mixed with the fluid formed in the Small-pox vesicles, which can scarcely be said to become pustules. There is great depression, but not often delirium—indeed, but rarely; the intellect usually remains clear to the last. A confluent eruption nearly always accompanies the malignant form of Small-pox, and death commonly takes place on the fifth day of eruption. *Petechial* Small-pox partakes very much of the same characters as malignant Small-pox. Numerous little dark spots, resembling flea-bites, especially about the armpits and groins, are observable, and the skin in these parts has a greenish yellow hue, very like what we see during recovery from a bruise. The condition of the fluids is no doubt very much the same in these two varieties of the disease; malignant and petechial Small-pox are very nearly akin.

6. *Variola Benigna.*—Van Swieten and others have described a form of natural Small-pox under the title of *Variola Verrucosa*, or *cornea*, stone-pock, horn-pock, and wort-pock, which we sometimes see in

these days, and in which the disease is of a mild, modified character. It is ushered in with symptoms as severe as in the dangerous confluent form, but on the third or fourth day of eruption all the severe symptoms begin to subside; the eruption assumes a modified form, such as we constantly see in post-vaccinal cases. We have often had an opportunity of observing this form of the disease in children whose mothers were at the hospital with them, and who knew perfectly well no attempt had been made at vaccination. The pustules are of unequal size, some shrivelling and dying off, while others are maturating; there is no secondary fever, and no pitting. These are examples of mild natural Small-pox, such as have occurred no doubt at all periods to a few favoured individuals, and in which, fortunately for the objects attacked, the disease leaves no trace behind.

7. *Variolæ Anomalæ.*—Small-pox is rendered irregular by being complicated with other diseases. We have seen it in conjunction with scarlatina,[1] measles, urticaria, syphilis, bronchitis, pneumonia, phthisis, dysentery, &c. Pregnancy may be mentioned as one of the anomalies; and another, the existence of Small-pox on the fœtus at birth; which must have gone through the stage of incubation, the primary fever, and early days of eruption, before it was born. We have several times seen children who were born with the eruption of Small-pox out on the body, but modified as it is on the mucous surfaces. Mead[2] imagined that persons who were insusceptible of Small-pox had possibly gone through the disease before birth.

First Symptoms of Small-pox, or Primary Fever.—This disease begins with rigors, fever, thirst, headache, sickness at the stomach, sometimes accompanied with vomiting, pain in the back, and general indisposition; followed, after forty-eight hours of illness, by an eruption on the skin of pimples, which are generally observed at first on the forehead, face, and wrists. Among the early symptoms of the disease should be enumerated, as now and then occurring in children, one or two convulsive fits. This occasionally happens in adults also, but not so often as in children. On passing the fingers over the points of eruption some hardness is felt in the skin, as if a grain of mustard-seed, or a small shot-corn, were embedded in it; but the skin is not tender to the touch at these points, nor does pressure seem to produce any pain. In cases of Small-pox after vaccination, which are so frequently met with in these days, the true or distinctive eruption of Small-pox is very often preceded by roseola, which lasts two or three days,—the *roseola exanthematica*,—which may lead observers, not intimately acquainted with the early symptoms of Small-pox, to suppose the patient has an attack of scarlatina; but this eruption may be known from that of scarlatina by not being so completely diffused over the skin as the rash of scarlatina usually is; it is also of a lighter, brighter, roseolar scarlet tint, than the eruption of scarlatina, which has a rather dingy hue; and, above all, it has a *mottled* appearance.

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[1] See Med.-Chir. Trans. vol. xxx. Marson, on the Co-existence of the Eruptive Fevers.

[2] De Variolis et Morbillis, cap. iv. edit. 1747.

Secondary Fever.—Besides the initiatory fever, the fever of invasion in Small-pox, there is what is called the secondary fever, which begins, in confluent cases, about the eighth or ninth day. In the milder cases of Small-pox, secondary fever is hardly perceptible; in the malignant and severely confluent cases death takes place before the secondary fever has barely commenced. But, in most instances of confluent Small-pox, patients suffer more or less from secondary fever, which seems to be the cause or forerunner of a very important chain of events. The pulse is increased in frequency, there is thirst, dry tongue, and hot skin; in many cases, particularly in the plethoric, some local inflammation arises, often occurring at the elbow, seemingly from leaning on it when taking food. But cellular inflammation takes place, in different patients, in nearly all parts of the body; sometimes it is deeply seated between the large muscles; twice we have known abscesses formed between the gastrocnemius and soleus muscles, causing intense pain during the formation of the matter. These deep-seated abscesses in Small-pox are sometimes the result of injury received months before. In one of the examples just mentioned, a seaman, in jumping from his ship to the wharf, missed his balance, and fell back on the ship, striking the calf of his leg against the side of the ship; no harm would most likely have followed had he not taken Small-pox or some other severe febrile disease.

Numerous small boils take place in many cases of confluent Small-pox; phlegmonous inflammation in others, involving often the greater portion of a limb. Patients who, from their previous good state of health, just escape dying from the severity of the eruption, at the usual time, viz. from the ninth to the thirteenth day, are very apt to suffer severely from secondary fever, and its consequences; such as pleurisy, pneumonia, ulceration of the cornea, &c.: these are amongst the very serious evils that may be expected. It is difficult to account for this peculiar form of fever in Small-pox; some have imagined it is owing to the absorption into the circulation of the pus formed on the surface of the body. If this were true, we should see, more frequently than we do, the results we recognise as belonging to pyæmia. These results we do see in some instances, but they may be said to be exceptional; whereas secondary fever is the usual consequence of confluent Small-pox. The absorption of some fluid forming part of the eruption, and more readily taken into the circulation than pus is, we strongly suspect to be the cause of secondary fever, but our knowledge of animal chemistry at the present day is not sufficiently precise to enable any one to say what this fluid is; it is one of the problems for the industrious and ingenious to solve. Secondary fever commences after, just after, the pustules have begun to discharge their contents; it may be that the absorbed fluid is not part of the original secretion, but the product of decomposition, or of some chemical change that takes place after the matter of Small-pox has been exposed to the air. In the horn-pock, as it is called from its hardness, in which there is some modification of the eruption in the advanced stage of the disease, such as we observe frequently after vaccination, the matter is dried up suddenly without being discharged at all: these cases are entirely free from secondary fever; therefore it would seem that the discharged matter has something to do with secondary fever.

Pleurisy is one of the most painful and fatal sequelæ of the secondary fever of confluent Small-pox. Patients are attacked with it very suddenly, and hardly ever recover; it runs a very rapid course, and terminates fatally in three or four days, sometimes sooner. The symptoms are generally at the first of a very decided character; violent pain in the side, wiry pulse, shortness of breathing, great difficulty in drawing the breath, and a very anxious expression of countenance. Such cases are all but hopeless; but we have, in some very rare instances, seen patients recover; they should, therefore, not be entirely given up as past hope; we had a very severe case lately in a young Scotchman, who, after a long convalescence, ultimately got well.

Pneumonia occasionally follows severe secondary fever. It comes on much more insidiously than pleurisy, and assumes the congestive character so well described by Mr. Erichsen, under the term of "Congestive Pneumonia," in *Med.-Chir. Trans.* vol. xxvi. p. 29. It is slower in its progress than pleurisy, and is very likely to have existed two or three days before it is discovered. It is more rarely, perhaps, seen than pleurisy, and is not so uniformly fatal; but it should be viewed as one of the very serious complications occurring in the advanced stage of Small-pox, and very likely to prove fatal.

Bronchitis is another serious complication of advanced Small-pox; dangerous at all times, doubly so when the body is weakened by other exhausting disease.

Glossitis sometimes arises during the secondary fever of Small-pox; the tongue becomes very much swollen and dry, so that the patient is unable to articulate or close the mouth; it is a very distressing and perilous symptom; those attacked with it nearly always die.

Otitis, followed by abscess in the ear, not unusually results from Small-pox. The pain produced during the formation of the matter is very great, but it is immediately relieved on the breaking of the abscess. It is probable that, in some instances, permanent injury may remain in the ear from this occurrence.

The abdomen escapes singularly free from complications in Small-pox. We do now and then meet with peritonitis, but very rarely; diarrhœa more frequently; and sometimes with mucous enteritis in children.

Erysipelas, pyæmia, gangrene, &c., are frequently met with, at times, in hospital practice. They form the most serious drawbacks to all hospitals, and are, as is well known, fatal in their tendency. These diseases are amongst the complications of Small-pox, and are not confined wholly to hospitals; they are met with in private practice, occurring after severe confluent Small-pox when the disease has lasted a fortnight or more. They are very dangerous. Erysipelas, more particularly of the head and face, occurs more frequently than any other form of superadded mischief. Patients with it, for the most part, get well; but erysipelas occasionally gives rise to pyæmia, which is followed by large abscesses, perhaps bed-sores, hectic fever, and death. The scrotum is apt to become gangrenous after Small-pox, especially in those who have the ill luck at the time to be suffering from gonorrhœa. It is a fatal complication; patients generally die who are attacked with it, but not always. We have several times seen the whole scrotum slough away, and the patient entirely recover; and it is interesting and surprising to see what a good covering is formed afterwards to the testicles, almost as good as before the scrotum was injured.

The women of the town are bad subjects for Small-pox. When they have gonorrhœa, they are very likely to have gangrene of the genitals; and, from their previous irregular habits and spirit drinking, their illness commonly ends fatally. We had a patient two years since with gangrene of the genitals, owing to leucorrhœa; it might, however, have been gonorrhœa; she was barmaid at a large hotel.

Variolous Ophthalmia and Corneal Ulceration. — Conjunctival inflammation often begins on the fifth or sixth day in Small-pox, and continues for a few days, and then subsides under the use of simple remedies. But there is another form of mischief—ulceration of the cornea—which often leads to the loss of an eye; both eyes, fortunately, being but rarely affected, although this does sometimes happen. Formerly, a large number of the inmates of the asylums for the blind had lost their eyes from Small-pox. The injury to the eye, by which the organ is destroyed, is not from the pustules of Small-pox forming on the eye, as used to be supposed, but from a destructive form of ulceration beginning almost invariably at the edge of the cornea.

After having been in constant attendance at the Small-pox Hospital for upwards of three years, and having witnessed the great epidemic of 1838, and having seen upwards of 1,500 cases of Small-pox, the author was induced to write a paper on Variolous Ophthalmia, which was read before the Westminster Medical Society in 1839.[1] Out of 1,500 cases, no instance had then, or for some time afterwards, come under his notice in which the pustule of Small-pox was formed on the eye. It does, however, happen now and then. In nearly thirty years the number of cases of Small-pox admitted into the hospital has exceeded 15,000. Out of this number 26 instances have been noticed in which the primary pustule of Small-pox has formed on the eye. It has not, however, in any one of these instances, injured the eye in any way; the cases have all done well.

In these very rare instances in which the pustule does form on the conjunctiva, it has nearly always been observed to have its seat halfway between the cornea and the inner canthus of the eye, where the conjunctiva is thicker than elsewhere. It has never been seen on the cornea. Now and then it has been observed half-way between the cornea and the outer canthus; the conjunctiva is thicker in this part also than over the eye generally. So that in these very exceptional instances, once in perhaps 500 cases, the pustule of Small-pox does form on the conjunctiva, but does not destroy or injure the eye in the least, so far as has been observed.

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[1] Medical Gazette, No. 32, May 4, 1839.

The ulceration of the cornea that leads to the destruction of the eye in Small-pox begins after the secondary fever has commenced. It has been observed to begin as early as the tenth day after the commencement of the general eruption, and as late as the thirtieth; the fourteenth day is a common time for it to be first seen. It comes on with redness and slight pain in the part affected, and very soon an ulcer is formed having its seat almost invariably at the margin of the cornea: this continues to spread with more or less rapidity, according to the degree of secondary fever present; in the more violent cases an ulcer being formed on each side of the cornea at the same time, showing the disease to be advancing with great severity, and presenting a tolerably certain indication that the eye will be entirely lost. The ulceration passes through the different layers of the cornea until the aqueous humour escapes; and if the part of the cornea destroyed be large, the iris protrudes through the opening. In the worst cases there is usually hypopyon, and when the matter is discharged the crystalline lens and vitreous humour escape; or the humours may escape from deep and extensive sloughing in the first instance, without the formation of matter; this being succeeded, of course, by the total annihilation of the form of the eye as well as the sight. In some instances the ulceration proceeds very rapidly, the entire cornea being swept away within forty-eight hours from the apparent commencement of the ulceration; and, what is singular, now and then the mischief goes on without the least pain to the patient, or his being aware that anything is amiss with his eye. This destructive ulceration never goes on rapidly but when there is a high degree of secondary fever present. That is a point which should be particularly remarked. It is likely to occur when there is a hot and dry state of skin, rapid pulse, thirst, loaded tongue; these having been preceded by a very confluent state of the disease, and the patient has just escaped dying at the usual time; namely, the ninth, tenth, or eleventh day of eruption. Then it is that some serious consequence may be apprehended, such as the loss of an eye, formation of large and deep abscesses, sloughing of the cellular membrane, or, may be, formation of matter in one side of the chest: some of these serious results may be expected when the secondary fever runs high in confluent Small-pox, combined with the circumstances above detailed.

It happens occasionally, unfortunately, that persons have had something amiss with their eyes before Small-pox comes on—some scrofulous tendency, or sensitive state of the conjunctival membrane caused by their occupation. For instance, a chimney-sweep was admitted into the hospital, and his eyes were in such a sensitive state from soot getting into them in the course of his work previously, that he had been several days in the hospital, keeping his head constantly under the bed-clothes, before he would allow his eyelids to be opened. When this was at last accomplished, both eyes were found to be entirely lost from ulceration of the corneæ.

Conjunctivitis, rather slow in its progress, begins in some cases during a tedious convalescence as late as the third or fourth week of Small-pox: after it has existed a few days, there will generally be found, on close examination, a small ulcer on the cornea; and, in this advanced stage of the complaint, the ulcer is commonly not at the margin of the cornea, but nearer the centre of it.

DIAGNOSIS.—It is often of great consequence to be enabled to decide as soon as possible on the nature of a febrile eruptive disease, as, in the cases of persons employed in large establishments, servants, &c., in order to their removal for the safety of others; and, on the other hand, it is unjust to the patients themselves to send them amongst Small-pox or fever patients, if they are not suffering from these respective diseases, where they may contract, and even die of a disease of a far more dangerous nature than the one they may happen already to be suffering from: on this account, early and correct diagnosis is of great consequence to all persons concerned, as well for the credit of the medical practitioner as for the safety of the patient. Upwards of twenty diseases have been mistaken, within the last few years, in the early stage of illness for Small-pox, and the patients have been sent as having Small-pox to the Small-pox Hospital. It has been observed, however, that three or four diseases mislead much more frequently than others; with the symptoms of these diseases, therefore, it will be desirable to contrast Small-pox. The four diseases are—Measles, febrile lichen, varicella, and some forms of continued fever. Some of the early symptoms of Small-pox are common to the other diseases above enumerated, such as fever, thirst, headache, sickness, and vomiting; but there is in Small-pox what there but rarely is in the other diseases—and when it exists it is accidental, not part of the disease itself—acute pain in the back, evidently not muscular pain.

1. *Diagnosis of Small-pox from Measles.*—Measles is far more frequently mistaken for Small-pox than any other disease is mistaken for it. In Small-pox the eruption follows on the *third day*, or after *forty-eight hours'* illness. In measles the eruption generally appears on the *fourth day*, or after *seventy-two hours'* illness; there is, besides, usually some cough, and lachrymal discharge and fiery redness of the eyes. The eruption, too, of measles, although a little elevated above the surface of the skin, is not so distinctly felt as in Small-pox; it appears more superficial. The lapse of forty-eight hours after the commencement of illness before the appearance of eruption, the pain in the back, and the shotty feel of the eruption on the skin in Small-pox, contrasted with the lapse of seventy-two hours of illness before erup-

tion in measles, the cough, redness of the eyes, and less marked feeling of hardness and prominence on the skin, should be enough, compared with the general appearance of the patient, to distinguish the two diseases.

2. *Diagnosis of Small-pox from Febrile Lichen.*—Febrile lichen is more *like* Small-pox, modified Small-pox especially, than any other form of disease is, not variolous. At first it must be confessed there is great difficulty in distinguishing between febrile lichen and modified Small-pox; still, however, by attending minutely to some leading characteristics, they may be distinguished: and here again *time* comes materially to our aid; lichen appears on the second day of illness, or after *twenty-four* hours' illness, and the eruption is without the order we observe in Small-pox; it appears scattered at random over the surface of the skin, and begins to appear at first generally on the trunk, as well as on the head and face, which is not the case in Small-pox. Two or three days will always, of course, put an end to any doubts there may be on the subject, as no fluid, or next to none, is ever found in the eruption produced in lichen.

3. *Diagnosis of Small-pox from Varicella Vera.*—Varicella vera leads to doubt in the minds of many practitioners. The distinction, however, between the two diseases is tolerably easy, and hardly ought to admit of mistakes. The initiatory fever of varicella is but very slight, scarcely perceptible; whereas it is generally rather severe in Small-pox, even where the resulting disease is mild. *Twenty-four hours only* elapse in varicella after the commencement of indisposition before the eruption begins to appear; there is no hardness, as in Small-pox, on passing the fingers over the points of eruption, and no areola at the base of each vesicle, or if any, very slight indeed; in most cases none. The eruption in varicella has its seat just under the cuticle, between the external and deeper layers of the epidermis; is vesicular, as if raised by a shower of boiling water; scattered over the skin without the order of threes and fives together, forming crescents and circles as in Small-pox; there is besides always, or nearly so, what is a very good guide, one or two large vesicles on the shoulders, generally between the shoulder-blades, much larger, and more spread out than the rest of the eruption, wanting the defined edge and hardness of the eruption of Small-pox. In the advanced stage the contents of the vesicles become purulent; but still, those who have watched the course of the disease carefully, cannot well be in doubt as to its real nature, and want of identity with Small-pox.

Even near the present day,[1] the doubt has not been altogether removed that varicella and variola may be of kindred origin. Heberden first pointed out clearly the distinction between the two diseases. We have no doubt whatever that they are quite independent of each other. Patients admitted with varicella into the Small-pox Hospital have often taken Small-pox during their stay there; and the converse happened a few years since; a child who had been in the hospital with Variola was discharged cured, and a short time afterwards was readmitted with varicella vera.

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[1] See Thomson on the Varioloid Epidemic of Scotland, 1820.

4. *Diagnosis of Small-pox from continued Fever.*—The slow insidious commencement of continued fever, with none of the suddenness and violence of attack observed in Small-pox, and the languid and general aspect of the patient in fever, ought always to be enough to mark the distinction between the two diseases, Small-pox and continued fever.

Prognosis.—In foretelling what will probably be the result of any particular case of Small-pox, the judgment should be guided by the most striking points already described. 1. The quantity of eruption; 2. The age of the patient; 3. Whether or not the mucous membrane of the larynx and trachea seems to be much implicated; 4. The state of the fluids giving rise to the malignant or petechial form of the disease; 5. The state of the nervous system, and previous habits of living; 6. Whether the patient has been vaccinated, and, if so, the number and quality of the cicatrices (to be alluded to particularly hereafter); 7. Whether the disease is complicated with pregnancy; 8. The favourable or unfavourable circumstances in which the patient is placed.

TABLE I.

Showing the rate per cent. of mortality from different forms of eruption in 2,654 unvaccinated cases of Small-pox, admitted into the Small-pox and Vaccination Hospital, London, from 1836 to 1851, inclusive.

Unvaccinated Small-pox.	Cases.	Deaths.	Rate per cent. of Mortality.
Confluent	1,838	937	50
Semiconfluent . . .	614	51	8
Distinct	202	8	4
	2,654	996	37

Note.—Eighty-one of the above patients who died were affected with antecedent, or superadded disease, as well as with Small-pox, viz. Confluent, 58; Semiconfluent, 15; Distinct, 8.

1. Confluent Small-pox is always more or less dangerous. Whenever the disease is confluent, the prognosis should always be very guarded in the early stage of the illness. Unvaccinated patients with this form of the disease die, as shown in Table I. at the rate of 50 per cent. Great confluence about the head and face is always to be dreaded, as patients often die with it when the eruption is but thinly scattered on the rest of the body. When the pustules are flat, do not acuminate well, and when the areolæ around them on the extremities are of a clarety hue, and the eruption on the face is white and of a pasty

appearance, the patient has but little chance of recovery. Distinct Small-pox is a disease of but little danger *per se* when uncomplicated with other symptoms of a fatal tendency; as shown in Table I. it produces a mortality of only 4 per cent.; it hardly ever alone endangers life in the adult.

Semiconfluent Small-pox produces, or is implicated in producing, twice the mortality of the distinct form of the disease; it is sometimes, in rare instances, accompanied with symptoms of malignancy, viz. hæmorrhage from the mucous surfaces, &c. and may become dangerous from the previous bad habits or shattered health of the patient. Under these circumstances a few deaths take place from semiconfluent Small-pox, amounting to 8 per cent.

TABLE II.

Ages of the unvaccinated patients admitted with Small-pox, at the Small-pox and Vaccination Hospital, London, from 1836 to 1851 inclusive, with the rate per cent. of mortality, calculated at different periods of life.

Date 1836 to 1851.	AGE IN YEARS.												Total.
	0–5	5–10	10–15	15–20	20–25	25–30	30–40	40–50	50–60	60–70	70–80	80–90	
Patients.	356	334	270	571	669	270	154	18	8	2	1	1	2,654
Deaths.	181	91	62	154	274	124	89	13	5	1	1	1	996
Percentage of Deaths.	50	27	23	26	40	45	57	69		75			37

Note.—About 2 per cent. of the unvaccinated patients died from Small-pox complicated with antecedent, or superadded diseases.

2. Age should occupy an important place in the prognosis of Small-pox. Its influence is the greatest in early and in advanced life. See Table II. 50 per cent. die under 5 years of age, and upwards of 50 per cent. beyond 30 years. The least mortality takes place from 10 to 15 years of life.

3. The state of the mucous membrane of the air-passages should be duly estimated; this can be pretty well known by the tone of the voice. When the larynx is much implicated there will be a good deal of cough, and the sound from coughing, and the voice in speaking, will have a hoarse metallic resonance. Laryngeal and tracheal complications render the disease very dangerous.

4. A knowledge of the condition of the fluids is very important in estimating the danger in Small-pox. All symptoms indicating malignancy, and a putrescent state of the blood, should be looked upon as very unfavourable signs. Hæmorrhage from any of the mucous surfaces, purpura, blood effused under the conjunctiva, or into the Small-pox vesicles, should all be regarded as very dangerous symptoms.

5. The state of the nervous system is amongst the most important points to be taken into consideration. Cases accompanied in the early stage with delirium generally end fatally. Persons of plethoric habit and free livers are very apt to have delirium, with a nervous, tremulous manner, and sleepless nights, and are very difficult to manage. The irritable temperament is unfavourable in Small-pox. Such persons often worry themselves about the merest trifles, and when they would otherwise do well, but for this irritability, the case ends in death. Delirium coming on for the first time about the tenth day is a very bad sign; such patients nearly always die. Children who grind their teeth hardly ever recover. The prognosis in the above cases should be unfavourable. On the other hand, a quiet state of the brain and nervous system, a tranquil cheerful manner, with hope of recovery, are tolerably certain indications of a favourable result.

6. The patient having been vaccinated will make a most important difference in estimating the danger from Small-pox. If the vaccination has been performed in four or more places, and corresponding cicatrices remain of good quality, readily seen, the case will, in all likelihood, end well. The early symptoms of Small-pox may be very severe, often are so, in well-vaccinated cases, but they subside as soon as the eruption is thrown out, which is usually highly modified, and all goes on well. But there are, unfortunately, many persons who have not had vaccination well performed, and they will suffer from Small-pox, probably, accordingly. When one or two cicatrices can but just be seen, doubtfully seen, the case may be as severe as if there had been no vaccination at all, the eruption pass through its several stages quite unmodified, and the disease proceed, and terminate, uninfluenced in any way by the previous vaccination.

7. Pregnancy is a most unfortunate and dangerous complication in Small-pox. Abortion is very apt to take place. In fatal cases the child is usually thrown off the day before death. It is generally born dead, but not invariably so. Although the danger in Small-pox is very much increased by pregnancy, and should always be taken seriously into account in forming a prognosis, pregnant patients occasionally do well, especially after vaccination. They sometimes abort, and sometimes do not; sometimes both mother and child do well.

8. The circumstances under which a person is placed in Small-pox may influence very much the result; as, for instance, on board ship; in a small, confined, ill-ventilated house; hospitals are especially dangerous to the pregnant woman, witness the mortality in the lying-in hospitals;[1] the prejudices of friends in overheating the patient, and giving cordials and strong drinks at unseasonable times: all these things may interfere with the chance of recovery.

SUSCEPTIBILITY TO SMALL-POX.—Each individual of the human species is born, it would seem, with a susceptibility to contract Small-pox, measles, scarlatina, and perhaps some other diseases, belonging to what is called the zymotic class—those diseases produced by a morbid animal poison. There is in the organism, most likely in the blood, some inborn principle or ingredient, clearly not essential to life and well-being, by which we are rendered liable to undergo these diseases. It is no doubt ordained by an overruling Providence that we should pass through these ordeals, from which hardly any are altogether proof, if they live but long enough, and from which large numbers annually die. After recovery from these diseases, the body is generally in no way better or worse for having passed through the change produced by the diseases, except in those instances in which the person is disfigured by the marks of Small-pox, or the seeds, perhaps, of scrofulous disease are brought into action; or, in the case of the measles, some pneumonic mischief may be left behind; or, in scarlatina, injury to the ears. All the functions essential to life usually go on as well as before, after passing through these diseases; therefore it would seem to be some innate principle in no way necessary to the well-being of the individual which is destroyed, or got rid of, during the attack; a principle or ingredient by which we are rendered liable to undergo these respective diseases. Some persons on exposure escape the infection of Small-pox over and over again, but take it at last. In 1844, a woman, 83 years of age, was admitted into the Small-pox Hospital with severe confluent natural Small-pox, of which she died, who had nursed her own children and her grandchildren with the disease, and had otherwise often been exposed to variolous infection, but never took it before. A similar instance is mentioned by Sir Thomas Watson, in his Lectures, of an old woman who had for years acted as a village nurse, and had nursed a great many persons with Small-pox, but at last, at 84 years of age, took the disease, of which she died.

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[1] See Lectures, by Dr. Barnes, in the Lancet, 1865, vol. i. p. 141.

Some persons have been known to pass through a long life, frequently exposed to Small-pox, but have never taken it; others, late in life, have taken it from inoculation, who had resisted taking the disease in the natural way, as it is called, namely, by breathing an infected atmosphere. Some few resisted, in inoculation days, both inoculation, and the natural mode of taking the disease; but these were very rare cases. All periods of life seem to be about equally susceptible to the influence of the contagion. In many parts of this country, before the invention of railways, Small-pox was absent for twenty years together. This happened more especially before the introduction of inoculation. Then, on the disease breaking out among the inhabitants of these but little frequented districts, the infection being conveyed to them by tramps, or dealers in small wares, the disease spread with fearful rapidity, and nearly all who came within the sphere of infection, whether young, or those more advanced in life, since the last invasion, took it, and it caused dreadful mortality, as it does in the present day, to the unvaccinated. All ages being taken together, it is found that about one-third, or rather more, of those who take Small-pox in the unprotected state—that is, who have never been vaccinated, or had Small-pox before—die of the disease. It is particularly destructive to the dark-skinned races; the blacks who come to the Small-pox Hospital suffer more from the disease than the native inhabitants of Great Britain. The same thing has been found to take place abroad. Dr. Bulkley, in the American edition of Dr. Gregory's "Lectures on the Eruptive Fevers,"[1] mentions an instance in which a tribe of American Indians took Small-pox, and they all died of it. Every individual of the tribe was swept away.

INFECTIOUS NATURE OF SMALL-POX.—Boerhaave was the first to point out the infectious nature of this disease. Before his time it was thought to depend on some peculiar influence of the atmosphere, and it is a remarkable circumstance that Sydenham, who paid so much attention to this disease, should have overlooked so obvious a property of it. Most likely it is communicable from the moment when the initiatory fever begins. It may be given by the breath of the patient before the eruption has appeared on the surface of the body. It continues infectious so long as any of the dry scabs resulting from the original eruption remain adherent to the body; a single breathing of the air where it is, is enough to give the disease. The dead body, for several days after death, has been known to communicate the disease (see Hawkins, in London Med. Gaz. vol. iii.); and in all probability it would produce the disease for some months afterwards. A few years since, a lady was walking at Islington, and met a person with Small-pox; twelve days afterwards she was taken ill, and for a few hours was delirious. The illness passed off without eruption. Her married sister, who had not been out of the house for three months, on account of pregnancy, was seized with illness exactly twelve days again after her sister's attack, which illness proved to be severely confluent but modified Small-pox. The case is singular and very interesting, as showing that the disease may be communicated by a person who had the early symptoms of the disease, precisely at the usual time after being exposed to Small-pox infection, but whose illness passed off without the characteristic eruption; a case in fact of *Variola sine eruptione* as first described by Sydenham.[2]

The infecting source bears no relation generally to the resulting disease; a mild case may, and often does give rise to a severe one; and, on the contrary, a severe case may produce a mild one. The dry scab of Small-pox would most likely set the disease going months, perhaps years, afterwards by inoculation, just as the dry scab of cow-pox has been found to be effectual for the purposes of vaccination after being kept a considerable time.

Clothes that have been worn by a person when suffering from Small-pox may retain the infection for a long time, as may the furniture, especially woollen furniture, of beds, and bedding, unless washed and thoroughly purified by exposure to the air, &c.

Recurrent Small-pox.—Small-pox but seldom occurs a second time. Instances of second attacks have, however, been recorded from the time of Rhazes to the present day. Thirty years ago we began to collect minute statistical information of all cases of Small-pox admitted into the Small-pox Hospital. At that time there were probably as many persons in this country who owed their protection to having been inoculated, or having had Small-pox, as to vaccination. We have communicated to the Royal Med.-Chir. Soc.[1] the particulars of this inquiry for sixteen years—1836 to 1851. Of 5,797 cases of Small-pox, 2,654, or 45 per cent. were unvaccinated; 47 cases, or less than 1 per cent. were after a previous attack of Small-pox or Small-pox inoculation; 3,094 cases, or 53 per cent. were after vaccination. It will, therefore, be seen that the cases of reputed Small-pox after Small-pox have been but comparatively few, and even some of these would perhaps admit of doubt; lichen, varicella, and some forms of pustular syphilis are difficult to distinguish from Variola, and might easily be mistaken for it, except by those intimately acquainted with the minute characteristics of eruptive diseases. The Small-pox Hospital has been founded 119 years, but there is no record of a patient having been admitted there twice, each time suffering from Small-pox. We have, however, no doubt of the disease occurring a second time, as measles and scarlatina do, but we think the instances are far more rare.

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[1] Page 34.

[2] Sydenham, vol. i. ch. 3, sec. 2.

An Irishman, the son of a medical officer in the army, who had been vaccinated in infancy by his father, and had a large cicatrix remaining from the vaccination, and who was attended by his father for Small-pox in early life, and bore decided pits of the disease, in 1844, at twenty-three years of age, was admitted into the Small-pox Hospital with severe confluent Small-pox, of which he died.

We have repeatedly seen the disease modified, when it takes place after natural Small-pox, or after inoculation, just as it is modified by vaccination. Among the circumstances that seem to predispose the constitution to receive a second attack of Small-pox is, as after vaccination, exposure for a time to great change of climate, either hot or cold. Women who have had Small-pox, or have been inoculated for it, often have, when suckling children with Small-pox, a few irregular spots formed on the breasts about the nipples; these spots are produced on the breasts by contact with the matter of Small-pox from the child's lips and face. There hardly ever is any accompanying indisposition; the effect is purely local, and cannot properly be considered to be a second attack of Small-pox.

Variola sine Eruptione.—Sydenham was the first to notice a form of fever without eruption, which prevailed at times when Small-pox was epidemic, and which he calls "*Variolous Fever.*"[2] "This fever originated in that particular epidemic constitution of the atmosphere, which, at the time in question, produced the Small-pox. Hence, with the exception only of those symptoms which were the necessary effects and consequences of the eruptions, it was, if not identical, at least closely akin to the Small-pox. Each disease set in similarly. In each there was the same pain upon pressure over the pit of the stomach. The colour of the tongue and the colour of the urine were alike in the two complaints. The profuseness and spontaneity of the sweats occurred equally at the commencement of both maladies. The common tendency to salivation was also equal. It occurred during the fever, when its heat and violence reached beyond a certain intensity. It occurred during the Small-pox, when the pustules became confluent. Finally, as the fever was most rife at that particular time when the ravages of Small-pox were greater in these parts than at any other time within the limits of my own observation, there can be but little doubt as to the identity of character between the two diseases. Of this I am certain—all those practical phenomena which determine treatment were the same for the two diseases, with the single exception of the eruption of the Small-pox, and of its effects."

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[1] Med.-Chir. Trans. vol. xxxvi.

[2] Sydenham, vol. i. ch. 3, sec. 2. Sydenham Soc. Edit.

De Haen has noticed a similar occurrence as having come under his observation. We have seen a few such cases that confirm the supposition. They occurred after vaccination, and are likely to be more numerous in these days than in the days of Sydenham, as we believe vaccination modifies Small-pox, in different persons, at every stage of its progress. Such cases are not likely to be sent in any large numbers to the hospital, as the eruption is the only decided evidence of the disease being Small-pox. Some like cases, occurring in a school, were reported to the Epidemiological Society, in 1852, in answer to a series of questions on Small-pox and vaccination extensively circulated among the medical profession.

M. Hedlund, giving an account of the Swedish epidemic of 1824, states (Magendie, Journal de Physiologie, tome vi.; and Gregory, Library of Medicine, vol. i. p. 303), "that three different forms of disease were then observed, all, as he believes, pathologically allied, viz. true Small-pox, the varioloid, and a fever without eruption. This fever, he adds, began and ended at the same time with the epidemic. The early symptoms were identical with those which preceded the variolous eruption. He considered it as a mild undeveloped Small-pox."

TREATMENT.—There is no specific for the cure of Small-pox. "It is a melancholy reflection," says Dr. Gregory, "but too true, that for many hundred years the efforts of physicians were rather exerted to thwart nature, and to add to the malignancy of the disease, than to aid her in her efforts. Blisters, heating alexipharmics, large bleedings, opiates, ointments, masks, and lotions to prevent pitting, were the great measures formerly pursued, not one of which can be recommended. What think you of a prince of the blood royal of England (John, the son of Edward the Second) being treated for Small-pox by being put into a bed surrounded with red hangings, covered with red blankets, and a red counterpane, gargling his throat with red mulberry wine, and sucking the red juice of pomegranates? Yet this was the boasted prescription of John of Gaddesden, who took no small credit to himself for bringing his royal patient safely through the disease. We may smile at this; but if either he, or Gordonius, or Gilbertus, were to rise from their graves and inquire whether this is one whit worse than Mesmerism, or at all more absurd than homœopathy, or hydropathy, we should, I fear, look a little foolish. Let us, then, avoid the errors of our ancestors, without reproaching them."[1]

One of the first things to arrange on undertaking to treat a case of Small-pox should be, if possible, to place the patient in a large airy apartment; bed-hangings, carpets, &c. had better be removed. The room should be kept cool in summer, and agreeably warm in winter, and the air of the room should be changed two or three times a day. In hospitals, the space allowed for Small-pox patients should not be less than two thousand cubical feet for each patient.

For a long time the custom was to keep patients with Small-pox as warm as possible; to heap bedclothes on them, to shut out every breath of air, forbid any ablution, or even change of body or bed linen. All this proceeding must have produced a horrible state of things. To Sydenham we are indebted—and a very great debt we may be sure we owe him—for having revolutionized all this. Like many other reformers of abuses, he was not able to accomplish the change he sought from these abuses without a good deal of obloquy. Thanks to his perseverance, he succeeded. We now use light bed-coverings, frequent change of linen, fresh air, ablutions, and cooling drinks, with the greatest benefit to our patients. To Sydenham we are also indebted for having first drawn the distinction between Small-pox and measles: no very great effort to accomplish, we should perhaps think, for any pathologist of the present day, the distinction seems so clear between the two; yet it was a step in pathology of great importance at the time, as Small-pox and measles had been for centuries looked upon as only modifications of the same disease; just as we, until quite lately,—thanks to the sagacity of Stewart and Jenner,—looked upon typhus and typhoid fevers as only modifications of the same fever. Like most other things, it seems easy enough to understand when once it has been clearly explained; but honour and praise are none the less due to the original observers. Easy as it seems, we might not have seen it; most likely we should not.

In the majority of instances it cannot be known for the first two or three days of Small-pox what febrile ailment is approaching; and, even if it were known, the mode of treatment would not materially differ. It will be right to give a dose of opening medicine to relieve the bowels, to keep the patient on simple diet, and to give saline medicine; nothing, generally, is better or more agreeable than citrate of potash, or tartrate of soda, in a state of effervescence. In confluent cases of Small-pox it is necessary to cut the hair close; in the unvaccinated, especially in children, the sooner it is done the better. But in the vaccinated exceptions should be made: to females, especially it is a great mortification to lose a fine head of hair, which will perhaps take two or more years to restore thoroughly; therefore it will be proper to wait until the *fifth* or *sixth* day of eruption to see if the course of the disease is modified, because, if it is, it will not be necessary to cut off the hair.

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[1] Lectures on Eruptive Fevers. Lect. V. p. 93. American Edition.

The diet of the patient should consist of tea and toast, without butter, bread and milk, sop, and oatmeal gruel; grapes, the *juice* of oranges, strawberries, and what patients are very fond of, and can have at all seasons of the year, roasted apples. For drink, toast-water, plain water,—which many prefer to anything else,—lemonade, imperial drink, milk and water, apple water, tamarind water, raspberry vinegar and water, and, what makes a very agreeable drink, some boiling water poured on black or red currant jelly. Sydenham says: "The moment that undoubted signs of Small-pox have shown themselves, I forbid the patient wine, meat, and the open air. His ordinary drink is weak small beer, with a toast put in to take the chill of. His food is oatmeal porridge, barley broth, roasted apples, and the like; articles which are neither hot nor cold, and which give no trouble to the digestion. I have no objection to a form of diet that is common in the country, and which consists of a roasted apple mashed with milk, only it must be taken at intervals, moderately, and with the chill off the milk. Hot regimen I forbid altogether. I forbid also all such cordials as are used by some under the rash notion of propelling the pustules towards the skin."

Fifty years ago, and later, it was not unusual to take away blood at the commencement of Small-pox: we never think of bleeding patients now at the Small-pox Hospital. Sydenham, Huxham, and others used to recommend bleeding in Small-pox, but, notwithstanding the sanction of their great names, it must always have been a very doubtful proceeding.

Delirium occurs in confluent Small-pox in persons of very different constitutions—most commonly, as previously stated, in persons of full habit, and free livers; but it also occurs in persons of weakly constitutions, and who may have lived temperately: the pulse is small and weak, and the features shrunken. Such persons require stimulants early—indeed it is about the only chance, doubtful as it is, of affording any assistance towards recovery. This form of delirium should, of course, be clearly distinguished, before giving stimulants, from the delirium of plethora.

Most writers on Small-pox allude to suppression and retention of urine as occurring in this disease; occasionally, perhaps not above once a year, we are told, at the Small-pox Hospital, a patient has not passed urine for several hours; but on examining the bladder there is not any distension of it. At the next visit, on inquiry, we always find urine has been passed. We have not had occasion to pass a catheter, in a case of Small-pox, for five-and-twenty years, therefore we conclude retention of urine must happen but very rarely in this disease.

An invariable complaint in Small-pox is soreness of the throat, more or less; this arises from the eruption being formed there as well as on the skin, which has to be explained to each patient, and it is necessary also to explain that we cannot stop the progress of the eruption there any more than on the skin, that it will go through a certain course in defiance of any means we may use to interrupt it. The spots necessarily cause more inconvenience in the throat, from the conformation of these parts, than on the surface of the body. All we can do for the relief of it is to recommend some mild gargle, or a small quantity of fluid to be taken frequently, or a little red or black currant jelly to keep the parts moist.

The bowels should be at once well cleared, at the commencement of the disease, by a dose, in the plethoric, of three or four grains of calomel, and eight grains of compound extract of colocynth, with or without sulphate of magnesia, and infusion of senna, and for the first few days they should be kept open two or three times a day. In the less robust, and in females, a salts and senna draught alone will, perhaps, do. Afterwards, in the course of the disease, if the bowels act daily without aperient medicine, all the better; if not, they should be relieved every two or three days by a salts and senna draught. So long as the tongue continues loaded with a brownish yellow fur, the salts and senna draught answers better than anything else; when the tongue is clean a dessert-spoonful of castor oil, or a rhubarb and magnesia draught, is more suitable; but so long as the tongue is clean, there is but little need for opening medicine at all; still the bowels should be relieved every few days. Not unfrequently it happens in Small-pox that the bowels are too much relaxed; for this we keep a mixture always in the ward of the hospital, and find it very serviceable :—

℞—Cretæ præparatæ,
Pulveris acaciæ
Sacchari albi, āā ʒiss.
Aquæ ℥iv.
Tincture opii ʒj.
Spiritûs ammoniæ aromaticæ,
Tincture catechu, āā ℥ss.
Aquæ menthæ piperitæ ℥iij. Misce.

Two or three table-spoonfuls a dose, to be repeated in *three hours*, whether the first dose seems to have answered the purpose or not; as without the second dose the diarrhœa will often return. If after three or four doses of the above chalk and laudanum mixture the diarrhœa still continues unchecked, having waited a suitable time, say three or four hours, it will be right to give three table-spoonfuls every four hours, of the compound infusion of roses; the sulphuric acid often answers the purpose of stopping the diarrhœa when the chalk mixture has failed; but the chalk mixture so generally affords relief that we always try it first. Should the two forms, above given, fail to stop the diarrhœa, ten grains of pulv. kino comp. may be given every six hours, or a scruple to half a drachm of the pulv. cretæ comp. cum opio. Rice and milk should be given as diet. If the above means all prove to be unsuccessful in stopping the diarrhœa, it will perhaps be found there is some tenderness, on pressure, of the abdomen; then a powder, or a pill, may be given every six hours, composed of three grains of hydr. cum cretâ, and two grains of pulv. ipecac. co.

In the early stage of Small-pox many patients are restless and unable to sleep at night; anodynes fail to procure rest. It may be worth while to try them once to see the effect, and repeat the dose or not as may be judged right. But there is in some patients the same wakefulness in the advanced stage of the disease, in patients who are otherwise doing well; then an anodyne given once or twice, just to get them into the habit of sleeping, answers admirably, and nothing does so well as the hydrochlorate of morphia: we have given it constantly for five-and-twenty years; it procures a comfortable sleep without causing thirst, or stupor, or confining the bowels, as tincture of opium does. It is convenient to keep a solution of it ready, four grains to the ounce: from twenty to thirty minims of the solution is a suitable dose; we generally find twenty-five minims to answer well.

One warning we are desirous of giving about the use of anodyne draughts: they should *not* be given when there is copious salivation and mucous expectoration. Patients at such times are very sleepless, because they require to be kept vigilant to discharge the saliva and viscid mucus frequently, almost constantly; if an anodyne be given under these circumstances, the patient goes to sleep, and the saliva and mucus, which ought to be frequently got rid of, go on accumulating during sleep in the air-passages, and thus the patient dies, gradually asphyxiated by the secretion accumulated in these parts.

Although the antiphlogistic treatment should be continued for perhaps the first few days after secondary fever has set in, patients shortly after its commencement require some additional support; beef-tea or calves'-feet jelly is very suitable to add to their diet, and a glass or two of the lighter wines may be allowed. The next step will be, supposing the patient to be going on pretty well, some soup, with a few shreds of thoroughly done meat in it. So long as the tongue continues furred, a meat dinner does not do well. Perhaps great weakness is complained of, and the appetite is bad; under these circumstances, a grain and a half of disulphate of quinine, with two or three minims of dilute sulphuric acid, and half a drachm of tincture of ginger in an ounce and a half of water, twice a day, will be serviceable. Game, poultry, or lightly boiled eggs might be allowed; and, in cases of great prostration, some brandy in gruel at night.

When the tongue has become clean, meat may be recommended, and some ale or porter, with or without wine, port or sherry; care being taken not to try to get on too fast.

Things, unfortunately, do not always go on so smoothly as this; some large collections of matter may form, with sloughing of the cellular membrane, requiring to be opened, or numerous boils harass the patient. It often happens that matter is formed under the scalp, small in amount at first, but it goes on collecting and spreading, and there is no disposition to point and break in this part as in other parts of the body. These collections should be *opened early* to prevent their spreading; the operation is rather painful, from the thickness of the scalp. A simple incision does not answer well; the matter collects over and over again, and the cavity of the abscess keeps getting larger; it is better done by a crucial incision, and the cavity should be filled with lint; these cavities are generally very tedious and troublesome in healing; nitrate of silver or a solution of it, freely applied to the interior, helps on the process. Instead of opening these abscesses by a crucial incision, a better plan, perhaps, to adopt is, to pass a seton through them, so that the matter may keep constantly draining away; we often resort to this proceeding with good effect, particularly when the abscess has been allowed to become rather large before anything is said about it.

Some form of steel, with or without quinine, is a useful medicine often, especially to females, during convalescence. Quinine and tinctura ferri sesquichloridi, or mist. ferri comp. should be tried. Cod-liver oil may sometimes be advantageously given, under such circumstances as it is otherwise found useful, in scrofulous subjects, or those inclined to phthisis.

The discharge from the pustules in some confluent cases is considerable and acrid; the itching, and discomfort produced by it on the skin, are relieved by the application of some absorbent powder freely used; flour applied with a common dredging box answers very well, or hair powder, starch, or calamine, dusted on the face, hands, inside of the shirt and sheets, will be found serviceable.

Many patients have numerous boils resulting from Small-pox; they leave ulcers which are tedious in healing; for some time no process of repair seems to be going on, and the discharge from the ulcers further exhausts the patient. Some decoction of bark, or quinine, with a few drops of dilute sulphuric acid, should be taken two or three times a day; and, in some instances, where the tongue is rather furred, two grains of blue pill, and three of compound extract of colocynth, may be usefully recommended every second or third night for a few times. The ulcers improve dressed with ung. elemi, or ceratum calaminæ; bits of lint, dipped in black wash, and applied to the wounds, and left on a couple of days, seem sometimes to do good. The majority of patients only require the wounds to be covered with bits of strapping.

Some patients like cold, others warm, applications in erysipelas; some prefer flour dredged over the inflamed part; collodion may be tried. If one plan does not make the patient tolerably comfortable, another should be tried. A liberal supply of wine should be allowed; the same in gangrene: both erysipelas and gangrene are generally preceded by bilious vomitings and very often by diarrhœa. During the sloughing of gangrene, at its commencement, nitric acid lotion, a drachm to a pint, may be used with benefit; later, some antiseptic should be applied to the part: one-third liquor calcis chloratæ to two-thirds water; or Condy's fluid properly diluted. Charcoal may be thickly applied, and covered with a linseed-meal poultice. A poultice made of beer grounds some recommend, others have a preference for carrot poultice; we generally trust to the solution of chloride of lime, or Condy's fluid.

When there is any gonorrhœal discharge from the genitals, either in the male or female, the bidet should be used, if possible, twice a day at the least, or some other means should be resorted to to keep the parts affected cleansed; if this be neglected, gangrene of the genitals is very likely to occur. The patient, unfortunately, is not well able at these times to use the bidet himself or herself, and the cleansing of these parts is a very unpleasant office for another to perform for them, and hence it is very likely to be neglected unless the medical attendant is very strict in enforcing his injunctions on the subject of cleanliness, and makes a point of inquiring daily if the bidet has been used.

Pleurisy is one of the most dangerous complications that can arise in the advanced stage of Small-pox. It soon, generally, carries off the patient. Bleeding is useless, if tried, and is, in fact, practically found to do more harm than good, and should be considered as inadmissible. The best plan to adopt—we believe, indeed, the only one we have seen to do good, and we have seen many tried—is at once to put a large blister on the side, and give a full opiate, forty minims of the solution of hydrochlorate of morphia, previously alluded to, or a like dose of tincture of opium; the dose to be repeated in twelve hours, unless the pain has very much subsided. Wine, if the patient has been taking any, had better, perhaps, be withdrawn.

Pneumonia, like pleurisy, arising in the advanced stage of Small-pox, in a person previously debilitated by an exhausting disease, does not admit of, and certainly will not be benefited by, any active treatment. Very likely, on carefully examining the chest, some consolidation of the lungs will be discovered. A blister should be applied, and five grains of blue pill given every night, or night and morning, for a few days; acetate of ammonia at intervals; an opiate at night, if very restless, and beef-tea as diet.

Bronchitis is another of the dangerous inflammations occasionally met with in the advanced stage of Small-pox, hardly ever admitting of anything but palliative treatment, yet likely to be fatal. Counter-irritation promises to be useful, and should be tried; and the inhalation of the steam of water, through a proper inhaler, always gives some relief. The lowering system, with repeated doses of calomel, &c. recommended by some writers, does not do well, and should be avoided.

Variolous ophthalmia, and ulceration of the cornea, are amongst the most serious results of confluent Small-pox. Bleeding, here again, used to be recommended, but the practice was bad; we soon saw it did a deal of harm, and was inadmissible. Quite the opposite mode of treatment, in our opinion, is indicated. The patient should be put on as generous a diet as can be borne, and allowed port wine, two or three glasses a day, and take quinine or liquor cinchonæ twice or thrice daily. To the eye, the following application may be made:—℞—Fot. papav. lb. j., pulv. aluminis ʒ j. pro fotu. Ung. cetacei to be applied each night between the eyelids. We sometimes touch the ulcer with nitrate of silver, scraped to a point. The eye should be fixed with a speculum—the one shown in Hey's Surgery answers well—and an assistant should be ready with some olive oil, in a grooved director, to drop into the eye, immediately after the caustic has been applied. Or the ulcer may be touched, by means of a camel's hair pencil, with a solution of nitrate of silver, a scruple to the ounce.

The conjunctiva, in the advanced stage of Small-pox, often as late as the third week, becomes inflamed, and after a few days a small ulcer may very likely be observed on the cornea. In such cases, as soon as observed, a blister to the temple is nearly always of decided benefit. Perhaps a second may be required—it often is. Should the conjunctival inflammation continue and the ulcer remain stationary, a solution of nitrate of silver, two grains to the ounce, may be dropped into the eye every second day, two or three times, with a large camel's hair pencil. Should the eye seem irritable, perhaps some vinum opii sine aromat. (to be had at Savory and Moore's) dropped within the lids, once or twice a day, may be serviceable. In nearly all cases, when lotions are not being applied to the eye, a green shade should be recommended to be worn. Scrofulous inflammation occurs after Small-pox, but it will be readily recognised by the great intolerance of light, and by its occurring chiefly in children.

From the earliest periods in the history of Small-pox strenuous efforts have been made to prevent the "pitting" that takes place from this disease. It must be confessed that it disfigures the countenance often terribly, and gives a very common expression to the handsomest face. We need not wonder then at the anxiety of friends, as well as of the patients themselves, that something should be done to prevent, as far as possible, future disfigurement. Some good can be effected, but when the disease is very severe the mischief arising from this cause cannot be wholly avoided. Velpeau recommended some years since that each vesicle should be opened and cauterized with a stick of nitrate of silver scraped to a point; to do good the operation should be performed on the *third* or *fourth* day of eruption. In the most confluent cases, those likely to produce the greatest disfigurement, the proceeding is scarcely practicable: it may be in semiconfluent cases. Mr. Higginbottom recommends the whole face to be washed with a strong solution of nitrate of silver, eight scruples to the ounce of water. We think this is much too strong, and that it will blister the whole surface; if used, half the strength will be enough. A mercurial plaster is used at the Children's Hospital in Paris, the form for which is given by Dr. Aitken;[1] it is a modification and simplification of the emplastrum vigo cum mercurio.[1] It consists of twenty-five parts of mercurial ointment, ten parts of yellow wax, six parts of black pitch. This application has good effect, but is most suitable for use in semiconfluent cases, or those barely confluent, where the patient can be prevailed on to use a little care in the management herself; in severely confluent cases the application would soon be rubbed off by the patient's restless movements.

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[1] Science and Practice of Medicine, vol. i. p. 263. Third Edition.

The application recommended by Dr. Graves, a few years since, of a solution of gutta-percha in chloroform, did no good, and by confining the discharge under the coating of gutta-percha, produced a most offensive condition of the patient. What we do generally is this—wait until the pustules have discharged, and the discharge has begun to dry, then put on some of the *best* olive oil, or a mixture of one-third glycerine and two-thirds of rose-water; some of this may be applied once or twice a day, for a few days, until the scabs begin to loosen. Cold cream and oxide of zinc, or olive oil and lime-water, form good applications; or if the discharge is thin and excoriating, calamine mixed with olive oil. The patient should be warned not to allow the scabs to dry and remain some time on the nose, and other parts of the face, particularly on the forehead and near the end of the nose; when this takes place, the dry scabs themselves leave deep marks in the skin, worse than the eruption of Small-pox itself. The pain of removing the dry scabs is sometimes considerable, and the patient can hardly be prevailed on to take them off, or allow others to do so. In common the pits from the eruption are not deep at first, just after the patient has got well, and we may deceive ourselves by thinking our efforts to prevent disfiguration have been attended with considerable success. The disease leaves a peculiar brown stain on the skin at first, which soon wears off, but the pitting is more perceptible a twelvemonth or so after the patient has got well.

One or two warm baths towards the end of the treatment should be enjoined in all cases of Small-pox.

MORTALITY FROM SMALL-POX.—Two circumstances, wholly different in kind, influence very much the mortality from Small-pox, as will be seen on referring to Tables I. and II. These circumstances are, the age of the patients, and the confluent form of the disease. Infancy and advanced age are unfavourable periods for undergoing Small-pox. Children under 5 years of age die at the rate of 50 per cent.; and adults above 30 years die in still larger proportions. See Table II. Patients, estimated at all ages, as they come to the Small-pox Hospital, die at the rate of 50 per cent. from confluent Small-pox; 8 per cent. from semiconfluent, and 4 per cent. from the distinct forms of the disease. See Table I. The most favourable time for taking Small-pox is from 10 to 15 years of age; beyond 60 years of age hardly any who take it escape dying.

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[1] See form in "Diseases of the Skin," by Erasmus Wilson, F.R.S. 4th Edit. p. 496.

Sydenham was fully aware of the dangerous day in Small-pox; the eleventh he says, which is the *ninth* day of *eruption*, as shown in the accompanying Table, No. III. The notion of the old authors of the critical days in Small-pox being the 7th, 14th, and 21st, is wholly wrong. The critical days, in fact, are really from the 8th to the 13th day, every one of these days being critical; but death may take place at any period, as seen in Table III.; the extremes being the 2d day in one instance, and the 168th in another. Patients may even die of the severity of the blood-poison from Small-pox before any eruption has appeared on the skin.

The following Table, No. III., formed from the Register of the Small-pox Hospital for 10 years—1855 to 1864—shows the days of eruption on which 987 cases proved fatal: by adding two days to any given number the period of illness may be known. Two-thirds of the fatal cases, it will be observed, took place during the second week of eruption:—

TABLE III.

Showing the days of eruption on which 528 unvaccinated, and 459 vaccinated cases proved fatal, from Small-pox, at the Small-pox Hospital, London, for the ten years 1855 to 1864 inclusive, and occurring amongst 1,537 unvaccinated, and 5,622 vaccinated cases. All patients having antecedent or superadded diseases of a fatal character have been excluded from the list, so as to represent the deaths from Small-pox alone, as accurately as possible.

	Unvaccinated Cases. Died on the			Vaccinated Cases. Died on the		
1st week	2d day of eruption	0	71	2d day of eruption	1	57
	3d	2		3d	2	
	4th	9		4th	7	
	5th	15		5th	7	
	6th	18		6th	17	
	7th	27		7th	23	
2d week	8th	53	350	8th	32	306
	9th	67		9th	50	
	10th	52		10th	57	
	11th	60		11th	64	
	12th	52		12th	39	
	13th	39		13th	28	
	14th	27		14th	36	
3d week	15th	21	67	15th	13	58
	16th	17		16th	12	
	17th	8		17th	8	
	18th	10		18th	7	
	19th	5		19th	8	
	20th	6		20th	5	
	21st	0		21st	5	
4th week	22d	2	21	22d	4	21
	23d	2		23d	1	
	24th	0		24th	4	
	25th	3		25th	4	
	26th	4		26th	4	
	27th	3		27th	3	
	28th	7		28th	1	
	Upwards of Four weeks		19	Upwards of Four weeks		17
			528			459

Morbid Appearances.—In all cases of death from Small-pox, the skin will exhibit, of course, different diseased appearances, according to the stage of illness at which the person has died. The most striking morbid results of internal parts are those displayed on opening the larynx and trachea, with its branches. These phenomena are peculiar to Small-pox. When the air-passages have been much affected by the disease, and when death has followed on the eighth or ninth day, the mucous membrane is found to be very much congested and inflamed, the epithelium in some instances separated, caused probably, Dr. Petzholdt thinks, by fluid effused between it and the mucous membrane, so as to produce vesication. It is also covered with a very viscid mucous secretion of a brown colour. After this is removed, the membrane appears thickened, pulpy, and in some instances ulcerated.

The next most remarkable morbid condition found in the dead from Small-pox, is the state of the chest after pleurisy. This can hardly be said to be peculiar to the disease, because a very similar state occurs after common inflammation. One side of the chest only is affected with variolous pleurisy; except, perhaps, in some very rare instances. The cavity of the chest is found filled with sero-purulent fluid; flakes of coagulable lymph floating in it; adhesions here and there between the pleura costalis and pleura pulmonalis, and the lung on that side of the chest rendered unusable from the pressure of the effused fluid.

Considerable difference of opinion has existed as to whether the pustules of Small-pox are ever to be found on the lining membrane of the alimentary canal. We do not believe that they are; at all events we have never found them there. If they ever exist, it is in some such rare cases as that reported by Dr. Patterson,[1] in which he believes he observed pustules on the mucous membrane of the colon. Sir Gilbert Blane, Rostan, and others have reported cases to a similar effect. The majority of writers on Small-pox believe that ulcerated spots on the mucous membrane of the intestines are due to other causes. Sir Thomas Watson says:[2]—"It is affirmed by some writers that the pustules of Small-pox occur in various internal parts of the body, and especially upon the mucous membrane of the intestinal canal. I believe this to be a mistake. The enlarged solitary follicles often put on very much the appearance of pustules."

"Many pathologists," writes Dr. Gregory,[3] "have expressed their belief that true variolous pustules have been found in the gastro-enteric mucous membrane. Others, again, among whom may be mentioned Cotunnius, Wrisberg, and Reil (who have paid great attention to the subject), are of opinion that this structure is incapable of developing variolous pustules, and that the appearances so described are in reality inflamed, enlarged, or ulcerated follicles, with petechial patches, similar in all respects to what are found in the common forms of idiopathic or typhoid fever. This pathological principle is fully borne out by the experience of the Small-pox Hospital. We may add, however, that even these appearances are very rare, and that the freedom of the abdominal viscera from urgent symptoms during life, and from all trace of disorganization after death, is a remarkable feature in the disorder. Inflammation may, indeed, originate from accidental causes in any internal organ during the progress of Small-pox, and its effects will be seen after death; but these are not to be confounded with the specific and acknowledged effects of the variolous poison upon the skin and mucous membranes of the throat and chest."

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[1] Edinburgh Monthly Journal of Med. Science, Feb. 1849, p. 549.

[2] Principles and Practice of Physic, 4th Edition, p. 862.

[3] Library of Medicine, vol. i. p. 308.

If vesicles of Small-pox ever are found on the gastro-intestinal mucous membrane, their course must be very similar to that described in the following quotation from Petzholdt, in the Brit. and For. Med. Rev. vol. v. p. 473: "There appear, on the lips and inner sides of the cheeks, small white spots, of a round or oval form, the centre of which is very frequently somewhat darker in colour. The epithelium is at these places much softened, and at length rises so as to form a small white vesicle, which is at no period transparent, the softened epithelium remaining always opaque and white; it is incapable of any great expansion by the fluid collecting beneath it, and soon bursts. The subjacent mucous membrane is to be seen at some points eroded on its surface. The course of such a pock is, consequently, very brief; the constantly moist state of the mouth rendering its actual filling with pus and desiccation, with the formation of a scab, altogether impossible."

When the lungs have become inflamed during or after the secondary fever of Small-pox, the morbid consequences will be found to be such as are observed after congestive pneumonia.

Bronchitis occurring during the progress of Small-pox leaves results similar to those seen after bronchitis generally. But we believe we have repeatedly observed, both during life and after death, the air-passages, larynx, trachea, and bronchi, all or some parts of them,—the larynx and trachea especially,—affected with *erysipelas*, leaving such morbid traces as might be expected after this low form of inflammation.

ANATOMICAL CHARACTERS OF THE VARIOLOUS POCK.—The variolous pock has been carefully examined with a view of describing its structure, by John Hunter, Dr. Adams, Petzholdt, Erasmus Wilson, Dr. Gustav Simon, and others. It is not so easy as might be supposed to fix on the exact spot where the variolous pock first begins to be formed. On examining a piece of skin of a person who has died on the third day of the eruption, there will be found to be patches of a whitish opaque substance between the epidermis and the true skin. These patches adhere firmly to the true skin, but, as Petzholdt says, they may be removed by syringing the part carefully with water, leaving the true skin nearly free from the new substance. If the epidermis, which includes the cuticle, properly so called, and the rete mucosum[1] "be examined when the eruption of Small-pox first breaks out, its undermost layers are found to be softened, almost spongy, and as if filled with a fluid. If a circular incision be made into the skin round the circumference of a papula, this being loosened by the cut from its lateral connexion with the skin, can be removed pretty easily with the pincers, in the form of a little knot. This experiment shows that, at the period of the disease in question, the connexion of the cuticle with the cutis is nearly destroyed at those parts of the skin which are affected, whilst a perpendicular section affords us a ready opportunity of satisfying ourselves that there is no cavity beneath the cuticle. During the growth of the pustules, the spongy softening of the cuticle is increased; a still greater quantity of fluid collects between the substance of its lowest layers; there at length arises a small cavity filled with fluid, and, by the increased accumulation of this fluid, the cuticle is gradually pushed upwards." "When the thin covering of a part of the skin occupied by a pock is removed, the cutis does not come immediately into view, but it is covered by a substance, varying in colour and consistence according to the degree of ripening of the pustule. At the time at which the formation of the cavity or hole described in the preceding paragraph commences, the fluid that covers the cutis is clear; at a later period it is turbid, more tenacious, and at length it becomes pure pus. If all these matters be removed, which is best done by a pretty strong stream of water from a small syringe, so as not to injure any of the subjacent parts, the following appearances can be seen with the aid of a microscope:—In all the pocks where pus has formed, there remains some of it behind, which cannot be washed off; and, if we employ for these investigations portions of skin that have had their vessels filled with red colouring matter, it can be seen with the naked eye that the pus is, as it were, wedged in between the bundles of vessels, and is retained by them and between them."

The depression in the pocks Dr. Petzholdt thinks is caused by the ducts of the cutaneous glands, which are ruptured as the pustules fill with pus and maturate, but which, in the early period of the eruption, bind down the cuticle to the cutaneous glands, and thus produce the pit or umbilicus.

Sir Thomas Watson says:[2] "Without going minutely into the anatomy of the pustules, you may distinctly see, if you closely examine them when they are five or six days old—you may see, at least, in many of them—two colours, viz. a central whitish disk of lymph, set in, or surrounded by, a circle of yellow puriform matter. In truth, there is, in the centre, a *vesicle*, which is distinct from the pus. You may puncture the vesicle, and empty it of its contents, without letting out any of the pus: or you may puncture the part containing the pus, and let *that* out, without evacuating the contents of the vesicle. The vesicles have even, by careful dissection, been taken out entire; and they are said to consist of several little cells. It is most probable that the lymph contained in this separate vesicle is the purest part of the variolous poison."

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[1] British and Foreign Med. Review, vol. v. p. 470.

[2] Principles and Practice of Physic, 4th Edition, p. 865.

Mr. Erasmus Wilson writes:[1] "When a well-formed and mature pustule is examined by dissection, it is found to be divided in its interior by a transverse septum into two chambers, both containing pus. The upper chamber is the larger of the two, and they communicate with each other, to a greater or less extent, by the rupture of the transverse septum around its marginal border. The epidermis forming the superficial boundary of the pustule, is the segment of a sphere, and continuous by its circumference with the cuticle covering the adjoining skin. The transverse septum is a layer of false membrane, of a whitish colour, which was deposited on the derma at an early stage of the pustule. Subsequently this layer becomes separated from the derma, and raised by the formation of pus beneath it, and at the same time it is broken around its edges, and permits the pus of the deeper cavity to communicate with that already contained in the superficial chamber. In consequence of the peculiarity in the mode of its production, this layer of false membrane generally retains permanently the umbilicated form of the primitive pustule, and is thinner at the centre than towards its circumference. When the septum is removed, the deep chamber is brought into view, and the depressed and sometimes ulcerated base of the pock exposed. The surface of the base is of a bright or purplish red colour, and highly vascular.

"Some difference of opinion subsists with regard to the cause of the umbilicated appearance of the pustule of Variola during its early stages. Dr. Heming many years since attributed it to the perforation of the pustule by the efferent duct of a sebiparous gland. Velpeau, who believes that the principal seat of Small-pox is the follicles of the derma, would, I suppose, entertain the same opinion. Other writers believe it to be produced by the pores of the skin, and Rayer refers it to the attachment of the false membrane. I agree with Velpeau that the follicles of the skin are the primary seat of the vascular congestion, that this congestion gives rise to the production of the papules or vari, and consequently that the epidermal sheath of the follicle is the probable cause of the umbilication of the Small-pox vesicle. When the vesicle is examined at its height of development, it is found to be multilocular in structure, and, when divided by a transverse section, exhibits an appearance which Gendrin has compared to a spice-box, while Bousquet likens it to a severed orange."

Dr. Gustav Simon[2] takes a different view of the subject: "I have found that variolous pustules are not always constituted entirely alike. In many cases where a central depression clearly existed, the epidermis was entirely raised from the subjacent cutis; and only at the spot corresponding with the umbilicus were both membranes united by a thin, whitish cord, which, as the microscope evidently showed, was a hair-sac. Upon the under surface of the epidermis, and for the most part also upon the upper surface of the cutis, was found a thin layer of a whitish mass, which, when looked at with the naked eye, possessed the characters indicated by Rayer;" which are, that "there exists in the Small-pox pustule, between the cutis and cuticle, a false membrane, which in shape resembles a truncated cone, and has a thickness of half a line." "The layer fixed to the epidermis was not connected with that lying upon the cutis; nor when the latter layer was absent, with the cutis itself; but the hair-sacs, ascending from the cutis to the epidermis, alone connected the two membranes."

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[1] On Diseases of the Skin, 4th Edition, p. 489.
[2] British and Foreign Med. Rev. vol. iii. 1849, p. 350.

"Other vesicles manifested a structure somewhat different from the above. In these also there appeared beneath the epidermis a white substance; but this, at the spot where externally the umbilicus was visible, adhered to the surface of the corium, so that the epidermis seemed fixed to the cutis by this white mass alone. Now as regards the white mass beneath the epidermic covering of the vesicles, and often also apparent upon the surface of the cutis, which most modern writers take for a false membrane, it consists, for the most part, of the deeper softened layers of the cuticle," &c. "As we are accustomed to give the name of false membranes to layers of coagulated fibrine, we cannot include the above-described white mass under this appellation; for, as I have before shown, the epidermic elements always form its major part."

"The fact that the described white mass, at certain spots of the vesicle, is prolonged uninterruptedly from the under surface of the epidermis to the cutis, while at other points this connexion is interrupted by the dissociation of the under layers of the epidermis, or by the complete separation of the cuticle from the cutis,—this fact, I repeat, is the occasion of the little divisions, or compartments, mentioned by most authors. These are usually of unequal magnitude and irregularly arranged; but sometimes I have seen them arranged with tolerable regularity. In cases of the latter kind, the white mass extended itself from the middle of the vesicle in the shape of little septa, like the rays of a circle, to the periphery, so that six or eight chambers of tolerably equal size were formed. In the middle of the vesicle, from which the irregularly arranged septa proceeded, a hair-sac was sometimes found. Frequently no separate compartments at all existed. In vesicles of this sort the epidermis was connected to the cutis in the middle by a thin white cord, or over a larger space; and around this centre ran a canal, into which the white mass extended, in the form of little clusters or lamellæ; but the canal was at no point interrupted by a complete partition. Rayer appears to have observed the same thing."

The reviewer says,[1] "That the production of an umbilicus is not dependent on the presence of a hair-sac or sebaceous follicle, is easily proved; for it is well known that neither exists in the hollow of the hand and foot: yet in children, in whom the thin epidermis permits it to appear, the central depression is frequently excellently marked. A remarkable peculiarity, first noticed by Rayer, characterises the variolous vesicle, formed beneath the horny cuticle, which, in the adult, invests the palm of the hand and the sole of the foot. On removing the epidermic cap of the vesicle and wiping off the fluid collected beneath, he observed that in the centre of the denuded corium a little elevation existed, while the circumference was visibly depressed below the level of the neighbouring healthy cutis. Dr. Simon has examined these points microscopically, and he finds that the central elevation consists of a file of papillæ of normal or nearly normal size and condition, while the depressed margin is paved with papillæ, bent down or flattened. The explanation is simple. At the centre of the vesicle, an organic connexion (from some yet unexplained cause) exists between the cutis and cuticle, and at this point no fluid is effused; but around this centre, exudation of fluid occurs without impediment, and tends to force the cutis and cuticle asunder. In other localities the cuticle yields, and rises everywhere, except at the umbilicus of the vesicle; but there the texture of the cuticle is so dense, and presents such great resistance to the distending power, that the cutis itself yields first, and sinks below the ordinary level." "Of the anatomy of pustules, Dr. Simon remarks, that the pus frequently exists between the cutis and cuticle, the process of suppuration commencing upon the surface of the cutis; but that frequently, also, the suppuration begins in the tissues of the cutis, extending thence beneath the epidermis."

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[1] British and Foreign Med.-Chir. Rev. vol. iii. p. 352.

The following is Wedl's view of the subject:[1] "External integuments.—Exudations in this situation are particularly fitted for study, being accessible to observation even during life. The most frequent are those which take place in the cutaneous *papillæ*, in which they are either confined to small limited districts, within which the exudation takes place around isolated groups of *papillæ*, or are more extensive. An instance of the former kind, or of a limited exudation, is afforded in Small-pox, in which the spots are at first filled with a limpid fluid, containing nothing but molecules, and do not become pustules till afterwards, when pus-corpuscles are developed in the hyaline exudation. The transudation takes place from the capillary system of the *papillæ*, the exudation as it is poured out gradually accumulating between the under surface of the *epidermis*, and the upper surface of the *corium*. But since the process is confined to limited groups of *papillæ*, the *epidermis* covering the latter is raised in the form of a transparent vesicle, whilst the spot at which the hair escapes from its sheath, together with the excretory duct of the sebaceous follicle, remains depressed, and constitutes the central *pit* of the vesicle. In those parts of the skin where no hairs nor sebaceous follicles exist, as in the palm of the hand and sole of the foot, the exudation deposited around a point where several of the deeper grooves in the *corium* meet, may cause a similar pit, since in a situation of this kind the *epidermis* constitutes a stronger layer, and is of closer texture. When the puriform fluid in the pustules begins to dry up, the pit becomes shallower and wider, owing to the subsidence of the swelling. In the integuments of a subject dead of Small-pox, it is easy to perceive that the vessels of the *papillæ* are more or less injected, when the skin has been macerated long enough to allow of the removal of the *epidermis*, beneath which the isolated patches of vascular injection are immediately apparent. Perpendicular sections show that the *papillæ* are the constant and principal seat of the injection; and it is from their vessels also that the hæmorrhage takes place, in cases of petechial Small-pox."

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[1] Rudiments of Pathological Histology, by Carl Wedl, M.D. Translated by George Busk, F.R.S. Sydenham Society, p. 206.

SMALL-POX AFTER VACCINATION.—VARICELLOID, AND MODIFIED SMALL-POX.

Vaccination was announced to the public, by Jenner, in 1798. The then prevalent practice of inoculation for Small-pox gave origin, no doubt, to the idea in the mind of Jenner, of performing a similar operation with vaccine lymph; coupled with the popular belief among the peasantry of Gloucestershire, that once having taken the disease from the cow, in milking these animals, such persons remained ever afterwards proof against the infection of Small-pox. Great hopes were entertained at first, and for some years, that all who availed themselves of vaccination would remain secure against Small-pox for the remainder of their lives. Independently of the popular belief that cow-pox afforded protection against Small-pox, it was thought by Jenner and the medical profession generally, that, as there was great similarity, if not identity, between vaccinia and Variola, and as Variola was believed to occur but seldom a second time, the same law it was imagined would govern the two diseases,—that those who had had the vaccine disease would remain ever after, or nearly so, secure against Small-pox; that they would enjoy the same immunity as those who had already had Small-pox, or at least as those who had been inoculated for this diseese.

Unfortunately this doctrine has not been altogether realized in practice. It has before been shown, p. 243, that at a time when there were probably about as many persons in England who had had Small-pox, or had been inoculated for Small-pox, as had been vaccinated, the numbers of those admitted into the Small-pox Hospital with Small-pox for a series of years, were but as 1 per cent. after Small-pox or inoculation, to 53 per cent. after vaccination. Notwithstanding this apparent drawback of vaccination from the first statements made about it, it was the greatest discovery in relation to disease ever made by man for the preservation of human life. The misfortune was that too much had been promised for it at first. Another great misfortune was, greater perhaps practically than the first, the public were taught to believe that the operation was so simple, and required so little knowledge and care, that anybody might perform it; and thus it has been in a great measure left to chance in England. It has never been taught by appointed teachers until five or six years ago, and even now it has not been taken up in a proper spirit, and treated by those in authority at the examining boards as it ought to be, and from its intrinsic importance really deserves to be. Nearly all that has been done in England, in regard to the more effectual performance of vaccination, and teaching of students, has been done at the suggestion and earnest entreaty of Mr. Simon, the able and zealous Medical Officer of the Privy Council; not, as we might reasonably have expected, by the Councils of our medical and surgical Corporations, who have the supervision and direction of medical education. Great care is given to teaching and learning the capital operations, as they are called, which not one practitioner in twenty through the whole country ever performs: no care, or next to none, to teaching and learning the other, which nearly all, when in practice, will have to perform frequently. Had certificates, fortunately, of having received instruction in vaccination been required of students at the examining boards, as they ought to have been, forty or fifty years since, hundreds of persons who have died, and thousands still living who have been badly vaccinated, and will still die of Small-pox after vaccination, might have been saved. As medical and surgical practitioners, our object should be to save all the lives we can by our art, no matter by what means; and if a little operation—little apparently in practice, but very important in its results—well performed can save many lives, as most certainly it can, and prevent much suffering and sorrow, it should surely always be done with the greatest care, and in the best known way. The success of all operations depends on nice care and management. Operations for hernia and for stone, for instance, if roughly, carelessly, and badly done, end badly; so it is with vaccination: and so far as the public are concerned, it is quite as objectionable to them, no doubt, to die of Small-pox because they have been carelessly and badly vaccinated, as it would be to them to die of hernia or stone because the operations for these complaints, respectively, had been badly performed. In the latter cases, the day of retribution would come immediately; in the former, unfortunately for its correction, it is delayed for perhaps twenty years, or more; otherwise, it would soon be set right. The operation, as an operation, has not been properly estimated from the first introduction of it in England, and it should be looked upon as a blot on our polity that vaccination has been worse performed, *generally*, in England, its birthplace, than in any other country in Europe.[1]

Some cases of Small-pox after vaccination were brought forward a few years after its first introduction into practice; and, in 1818, a work appeared by Dr. Monro, of Edinburgh,[2] treating particularly on the subject. In 1819, nineteen cases of Small-pox after vaccination were admitted into the Small-pox Hospital, London, according to the Report of Dr. Ashburner, then physician to the hospital, to the Court of Governors of the hospital held at the end of that year. In 1820 the work of Dr. John Thomson[1] was published, describing the disease which had lately prevailed in Scotland, and naming it a "Varioloid Epidemic." In 1824 Small-pox after vaccination prevailed to a great extent in Sweden. In 1825 Small-pox was epidemic in London, and attacked several persons who had been vaccinated, 147 of whom were admitted in that year into the Small-pox Hospital. In 1828 there was a severe epidemic of Small-pox in Marseilles, when about 2,000 were attacked who had been vaccinated. Between July 1831 and June 1836, as reported by Dr. Heim, 955 persons were attacked with Small-pox after vaccination in the kingdom of Wirtemberg, of whom 75 died, or 7·8 per cent. From this period cases of Small-pox after vaccination have kept gradually increasing in numbers, until they now amount to four-fifths of the admissions into the Small-pox Hospital.

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[1] See Med.-Chir. Trans. vol. xxxvi. p. 381.

[2] Observations on the different Kinds of Small-pox, and especially on that which sometimes follows Vaccination. Edinburgh, 1818.

The introduction of vaccination has rendered the diagnosis and course of Small-pox, and consequently the study of the disease, far more intricate than it used to be in former days, and now is in the unvaccinated. The phases of the disease have been made by vaccination far more numerous than they were before. A large majority of the cases of Small-pox occur at the present time (1865) after vaccination. Thirty years since, from 1835 to 1845, the admissions of patients into the Small-pox Hospital, were 44 *per cent.* of Small-pox *after vaccination;* from 1845 to 1855, 64 *per cent.;* from 1855 to 1865, 78 *per cent.*, and during the last two years (1863-4), 83 and 84 *per cent.* respectively. It will therefore be seen that they are gradually increasing in numbers; and, now that vaccination has been made by law compulsory in England, the percentage of cases of Small-pox after vaccination will no doubt go on increasing, until it will be rather a rare circumstance to see Small-pox in the unvaccinated, at least in the adult. Unfortunately, the number of admissions into the hospital keeps also increasing, owing principally, doubtless, to the rapid increase of the population of the metropolis, thus:—

Admissions into the Small-pox Hospital, 1835 to 1845 — 3,494
,, ,, 1845 ,, 1855 — 4,546
,, ,, 1865 ,, 1865 — 7,326

Now there can be no doubt that what has been observed for the last thirty years at the Small-pox Hospital, London, in relation to the occurrence of Small-pox after vaccination, has been going on, in much the same way, all over the country, wherever Small-pox has prevailed.[2]

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[1] An Account of the Varioloid Epidemic, which has lately prevailed in Edinburgh and other parts of Scotland, 1820.

[2] See Report of the Small-pox and Vaccination Committee of the Epidemiological Society, ably drawn up by Edward Cator Seaton, M.D. Hon. Sec. to the Committee, 1853. Also a valuable contribution by the same author, "On the Protective and Modifying Powers of Vaccination,"—a pamphlet, reprinted from the Journal of Public Health and Sanitary Review, 1857.

The disease, as modified by vaccination, received the name of "varioloid," first suggested for it by Dr. John Thomson, of Edinburgh, who wrote a work on the subject in 1820.[1] This word "varioloid" has been adopted generally throughout Europe by writers on Small-pox—in France, Germany, Denmark, &c. &c.—in treating of Small-pox as modified by vaccination. It appears to us not to have been well chosen, inasmuch as the disease is not simply "like" Small-pox, as the name implies; it *is* Small-pox: it will give the disease in the most severe form, in the natural way, by infection, to the unvaccinated, and will produce Small-pox by inoculation just as a case of Small-pox uninfluenced by vaccination will do. There is not a good name, perhaps, for it at present. When the disease is highly influenced, and altered in its course, rendered mild, by vaccination, we call it at the Small-pox Hospital, *Variola Varicelloides*—Variola like Varicella, which is a better term than "varioloid," the meaning of which is simply, "like Variola;" not a very satisfactory way of explaining what is meant. Perhaps the best term for it is, "Small-pox modified by Vaccination," when it is modified; but it is not always modified in persons who have been vaccinated. Besides, there are various degrees of modification, when it is modified; it is not always even like Varicella unfortunately, but often hardly modified at all, and such patients frequently die. Small-pox after vaccination has, in fact, various degrees of severity and modification, from the slightest form in which there is none, or hardly any eruption at all, to the most severe confluent cases, closely, often exactly, resembling the disease in the unvaccinated; and it also assumes the petechial and malignant types after vaccination just as in the unvaccinated state. All this depends in a great measure on the way in which patients have been vaccinated. Those who have been fortunate enough to have been vaccinated in four or more places with lymph that leaves good, easily perceptible cicatrices, have almost invariably a slight form of Small-pox when it occurs; but those who have only one or two marks from vaccination, such as are hardly visible, will probably have a severe form of the disease; and those who have no marks at all are in still worse circumstances. Now, although this rule holds good *generally*, almost *invariably*, still it is not an invariable rule; and perhaps more exceptions will be found—we may say will certainly be found—in those who have been indifferently vaccinated, than in those who have been well vaccinated. Persons seemingly indifferently vaccinated will oftener afterwards have a light form of Small-pox than well-vaccinated persons will have a severe form of the disease. So far it is fortunate. But what we contend for, and always have contended for, is that, if possible, all should be vaccinated in the *best way;* at least the attempt should be made to vaccinate all in the best way, that there should be as little as possible of hap-hazard vaccination, done with a view that if the operation takes effect badly it can be done again. By such a proceeding persons often take vaccination badly, and cannot be made to take it properly afterwards; the imperfect success prevents its taking fully again, and yet some day they may take Small-pox severely, and perhaps die of it. Every effort should therefore be made that there may be as few imperfect vaccinations as possible.

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[1] An Account of the Varioloid Epidemic of Scotland.

It is, however, undeniable that a few cases occur even to the best vaccinators that do not take the vaccination well. Children sometimes fall ill from other causes just after being vaccinated. There cannot always be good subjects at hand to vaccinate from; the weather and other circumstances interfere with the regular attendance of those who have been vaccinated to afford supplies of lymph for the vaccination of others; but careless and bad vaccinators avail themselves of these untoward circumstances as an excuse for having frequent failures. Great care and great nicety of management are requisite for uniform or almost uniform success in vaccinating, and without the observance of this nice care and management many cases of vaccination do badly, or do not take at all: after two or three failures the friends of a child are led to think it cannot be got to take the vaccination, and they neglect to have further trials made, and ultimately the child takes Small-pox, perhaps severely; such results are of frequent occurrence.

TABLE IV.

Analysis of all the cases of Small-pox after Vaccination, admitted at the Small-pox and Vaccination Hospital, London, for a period of 20 years, viz. from 1836 to 1855, inclusive, showing, from a careful examination of the cicatrices, the relative amount of security given by the number of vesicles produced at vaccination; and, judging from the character of the cicatrices, the probable state of activity and efficacy of the lymph used for Vaccination.

Patients admitted with Small-pox.	Number of Patients.	Character of Cicatrices.	Cases.	Results.			
				Discharged.	Died.	Died affected by superadded disease.	Rate per cent. of Mortality from Small-pox, after deducting entirely the Cases affected by superadded disease.
1. Having one vaccine cicatrix . . .	2001	good	1032	978	54	15	3·83 } 7·73
		indifferent	969	835	134	21	11·91
2. Having two vaccine cicatrices . .	1446	good	873	841	32	12	2·32 } 4·70
		indifferent	573	516	57	10	8·34
3. Having three vaccine cicatrices. .	518	good	307	300	7	4	0·99 } 1·95
		indifferent	211	202	9	2	3·34
4. Having four or more vaccine cicatrices	544	good	358	356	2	0	0·55 } 0·55
		indifferent	186	183	3	2	0·54
5. Stated to have been vaccinated, but having no cicatrix . . .	370	—	370	269	101	18	23·57
6. Stated to have been vaccinated, but particulars of cicatrix not recorded	17	—	17	14	3	2	6·66
	4896		4896	4494	402	86	6·56

Notes.—A good vaccine cicatrix may be described as distinct, foveated, dotted or indented, in some instances radiated, and having a well, or tolerably well, defined edge.

An indifferent cicatrix—as indistinct, smooth, without indentation, and with an irregular and ill-defined edge.

Aggregate mortality with *good* vaccine cicatrices, from Small-pox alone, uninfluenced by other diseases, 2·52 per cent.

Aggregate mortality with *indifferent* vaccine cicatrices, from Small-pox alone, uninfluenced by other diseases, 8·82 per cent.

There is found to be a mortality of about 2 per cent. in vaccinated as well as unvaccinated patients, from Small-pox being complicated with antecedent or superadded diseases.

From the foregoing Table, No. IV., it will be seen that nearly five thousand cases of Small-pox after vaccination are reported on, each case having been carefully recorded at the time in the hospital register, and the whole subsequently arranged under different headings, so as to show the value of the different modes of vaccinating, in persons who have had Small-pox after having been vaccinated several years previously.

Three-fourths of the cases had taken the vaccination in but one or two places, and among these, by far the largest proportionate mortality from Small-pox has fallen; and what should also be strongly impressed upon the memory is, that by far the largest amount of suffering and disfigurement have been produced in those in this category who recovered. It is not simply those who died in the one class of cases that suffered more than others to which we are desirous of drawing the attention, but to those belonging to this class who escaped dying that also suffered and were disfigured, a great deal more than those in another class in whom the vaccination had taken effect in four or more places and recovered. This is very important to keep in mind. By vaccinating so as to take effect in four or more places, we not only save life, but prevent a great deal of suffering and subsequent damage to the appearance of the person, which in females, at least, is of great consequence, and not always quite a matter of indifference to males.

It will be observed that of 544 cases having four or more vaccine cicatrices, only half of 1 per cent. died of Small-pox, or 1 in 200; whereas of 969 cases, with only one indifferent vaccine cicatrix, just upon 12 per cent. died; in each instance antecedent or superadded disease has been deducted, so as to leave the death, as far as could be known, purely the result of Small-pox. Here, then, is a very important practical point to bear in mind when vaccinating: if only one indifferent cicatrix remains from the operation, such persons, taking Small-pox in after life, die at the rate of 12 per cent.; on the other hand, if four or more cicatrices remain, only half of 1 per cent. will die of Small-pox.

It should be further observed that, of 370 persons who believed themselves vaccinated, but who had no cicatrix to show for having been vaccinated, but who trusted to it for their protection, they died of Small-pox at the rate of 23½ per cent. This again is a very important practical point to bear in mind; persons having no cicatrix remaining from vaccination should by all means be urged to be re-vaccinated, else they may very likely some day fall a prey to Small-pox; such persons are in a very unsafe position.

Now, what has been going on in London for years past, as seen in patients who have been admitted with Small-pox at the hospital, has been going on also, there can be no doubt, all over the country, in persons who have gone through the disease elsewhere—a very large majority of the patients admitted at the hospital are not Londoners, but persons who have come to London from the country, to serve in the capacity of servants, or are employed in different ways of business. They were vaccinated in the country.

The security of vaccinated persons will be seen, by Table IV., gradually to rise, not only from the number of cicatrices produced at vaccination, but also according to the *quality* of the cicatrices, agreeing with the description given, in the notes of the Table, of the characteristics of good and indifferent cicatrices. Active vaccine lymph, such as leaves clear permanent cicatrices, is evidently indicated as the most desirable to select for use in vaccinating. Another recommendation is, that it takes effect more readily at the time of using it. Long humanized lymph requires a more dexterous hand to produce anything like uniform and perfect success.

In the notes of Table IV. it will be seen that the aggregate mortality in persons who have Small-pox is much less in those with good, than in those with indifferent vaccine cicatrices: thus taking all the cases together, regardless of the number of cicatrices, those with *good* cicatrices died at the rate of 2½ per cent. only; whereas those with *indifferent* cicatrices died at the rate of 8¾ per cent.: this is also a very important subject to remember, carrying with it, as it does, the increased amount of danger and suffering when Small-pox arises. One really good, circular, radiated, and indented cicatrix, is worth two or three indifferent cicatrices generally, such as can hardly be seen. With good, active, eighth-day lymph, an expert vaccinator will hardly ever fail; certainly not above once in 150 times. We may, indeed, confidently state, from those on whom we can rely, and from our own experience in vaccinating, that the failures will not be nearly so often as once in 150 times; they should be very rare occurrences indeed, in good hands.

In the course of years, vaccine lymph becomes humanized, by passing many times through the subject, and can only be kept in a good state of efficiency by having many subjects constantly to select from for its continuance, and even then the cicatrices it leaves, after many years' use, are not so good as they were formerly: this is a point on which we can bear witness from our own experience, and it is shown by the above Table how very important it is to have lymph that leaves good permanent cicatrices.

Out of large numbers of cases of Small-pox after vaccination, viz. 1,958, admitted into the Small-pox Hospital during the years 1863 and 1864—Small-pox having been epidemic in London throughout these entire years—the mortality after vaccination shows a considerable increase, viz. from 6·56 per cent. as given in Table IV., for twenty years—1836 to 1855—to a mortality of 9·2 per cent. out of 1,958 cases, for the years 1863-4; all patients manifesting antecedent or superadded diseases having been deducted from the calculation, as in the former instance, in making Table IV.

It is a question that may be fairly and properly entertained, and deserves very mature deliberation, whether we ought not to resort more frequently than has hitherto been done to supplies of lymph from the cow. A good stock should not be carelessly given up; with many subjects constantly to operate on, and choose from, it can be kept for many years in a good active state of efficiency; and, however many may be vaccinated, the lymph can only make the circuit of the human body fifty-two times every year, yet this multiplied by twenty or thirty, as the case may be, according to the number of years, makes the total number considerable. The able and interesting researches of Mr. Ceely of Aylesbury,[1] by which he established the practicability of obtaining lymph from the cow, by inoculating these animals with the virus of human Small-pox, has secured to us the means of procuring fresh supplies of vaccine lymph should it be difficult to meet with the disease in its natural state in these animals.

Mr. Badcock, formerly of Brighton, now of Camberwell Grove, informs me that he has succeeded thirty-seven times during the last twenty-five years, in inoculating the cow with the virus of human Small-pox.

The subject of deterioration of vaccine lymph will be of course a disputed point. It will, perhaps, be argued that diseases of this class do not alter; that they remain perpetually the same; that Small-pox, measles, scarlatina, &c. are the same now as they were originally, and ever will be. This argument may be granted as true generally, but it should be remembered that vaccinia is not genuine Variola, even when we know the disease has been produced in the cow by inoculation with the virus of human Variola. In passing the disease through the cow it thereby undergoes alteration, and is not afterwards communicable to man by infection as true Variola is; nor is vaccinia one of the diseases natural to man, but is only taken by him through the agency of inoculation: therefore, on this account, it is likely from a variety of circumstances to undergo degeneration, as it is a disease produced at will—artificially—not taken when the body is most prone to receive it, as we may suppose the body is when disease is taken in the natural way. We feel bound, however, to state we have frequently produced, lately, with lymph brought into use by Jenner more than fifty years since, vaccine vesicles which, on comparison, exactly correspond with the vesicles sketched in Jenner's original work, explaining and illustrating the vaccine disease; but we also feel bound to state for consideration, reflection, and practical deduction, the facts with regard to Small-pox after vaccination which have come before us at the Small-pox Hospital, for the last thirty years.

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[1] Observations on the Variolæ Vaccinæ, and Variolation of Cows, by Robert Ceely, Esq. Surgeon to the Buckinghamshire Infirmary. In Transactions of the Provincial Medical and Surgical Association, 1840.

According to a statement of Dr. Sanderson,[1] as reported to him by the local medical practitioners, the cows still suffer from the true cow-pox every spring, or nearly so, in the neighbourhood of Berkeley, Gloucestershire, the birth-place of Jenner.

Every now and then a patient is admitted into the Small-pox Hospital who has been vaccinated *after* the symptoms of Small-pox have appeared, the disease being known to be Small-pox when the vaccination was performed. Several patients were thus admitted a few years since from one of our largest west-end London parishes. These vaccinations must have been done under a total misapprehension of the powers of vaccination to control Small-pox. It does no good whatever when so performed, and should not be repeated, as it only tends to bring vaccination into discredit with the public. The facts of the case are these:—Vaccination to be effective should have gone on to the stage of areola before there is any illness from Small-pox. It has before been stated that when Small-pox has been taken into the system there is twelve days' freedom from illness generally, forty-eight hours' illness, and then the disease begins to appear on the skin. The areola of vaccination is not fully formed until the ninth or tenth day of the progress of the vaccine vesicles, on those who have never been vaccinated before; so that unless there has been time for the areola to be formed after the vaccination, before the illness produced by Small-pox begins, the vaccination will not be of the least benefit. The progress of vaccination is generally—not always—interrupted as soon as the illness from Small-pox commences, but as it never can be exactly known when Small-pox is taken, so long as persons are well in a house where it exists it will be right to vaccinate or re-vaccinate, as the case may be, as soon as possible, all likely to contract the disease; but this is a very different thing to vaccinating *after* the symptoms of Small-pox have actually commenced. Example:—Suppose an unvaccinated person to inhale the germ of Variola on a Monday: if he be vaccinated as late as on the following Wednesday, the vaccination will be in time to prevent Small-pox being developed; if it be put off until Thursday, the Small-pox will appear, but will be modified; if the vaccination be delayed until Friday, it will be of no use, it will not have had time to reach the stage of areola, the index of safety, before the illness of Small-pox begins: this we have seen over and over again, and know it to be the exact state of the question. Re-vaccination will have effect two days later than will vaccination that is performed for the first time, because re-vaccinated cases reach the stage of areola two or three days sooner than in those persons vaccinated for the first time. Four or five years since, Small-pox occurred in a family at Richmond, consisting of a man and his wife, and their niece. We received the account of the occurrence from the man, the only one of the three admitted into the Hospital. The niece was the first attacked with the disease, and died. About a fortnight afterwards the woman was attacked, and died; both at Richmond. During the woman's illness the man was vaccinated, and he had five large vaccine vesicles on his arm, *without any areola,* on his admission, in the early stage of Small-pox, at the hospital. The progress of the vaccine vesicles was arrested by the Small-pox; the man went through confluent Small-pox, wholly unmodified, and died. Now had the vaccination been performed one day sooner, the Small-pox would have been modified—two days sooner, and the man would have been saved from it altogether: this shows the necessity of performing the vaccination as promptly as possible in houses where Small-pox exists. Three weeks or a month had been lost after the first outbreak of Small-pox in the house before the vaccination was performed on this man.

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[1] Sixth Report of the Medical Officer of the Privy Council, with Appendix, 1863, p. 213.

Re-vaccination.—It is found on examining large numbers of persons attacked with Small-pox after vaccination, that the majority of those attacked are from eighteen to twenty-five years old, and that they had been almost invariably vaccinated in infancy.[1] This may be partly accounted for, no doubt, by persons of this age coming to London from the country to act as servants, in shops, &c. But it would seem, that all persons at this age become more liable to take the disease then than earlier, or later, in life; they become, in fact, more susceptible to it at this age than sooner after vaccination. It would, therefore, seem to be a wise course to pursue to recommend all persons on reaching adult age, especially if about to change their place of residence, to be examined as to their probable security against Small-pox. If they have four or more good cicatrices from vaccination, they are tolerably safe; if, on the other hand, they have but one cicatrix, and that such as can hardly be seen, or no cicatrix at all, such persons had better be re-vaccinated as a matter of precaution. These remarks apply especially to persons on passing from one part of the world to another, more particularly if the climate be very different from the one where they have been living. Any change from either a hot to a cold, or colder climate, renders persons liable to contract Small-pox. Persons coming from India to England, should be re-vaccinated. Vaccination, judging from the cicatrices, does not appear to take effect so well in India as in England. At certain seasons of the year there, during the hot season, the vaccine lymph is said to suffer deterioration. However this may be, on examining persons who have been vaccinated in India, the cicatrices left by vaccination will generally be found to be very indifferent. Further, it would seem, that persons who have been inoculated for Small-pox or have had Small-pox, are liable to be attacked with this disease a second time, under the circumstances mentioned, and that a predisposition to contract the disease is renewed by any great change of climate, either to a hotter or colder temperature.

In commendation of re-vaccination we may state that but very few patients have been admitted with Small-pox into the Small-pox Hospital, who stated they had been re-vaccinated with effect, and that these few have had Small-pox in a very mild form. For just upon thirty years we have re-vaccinated all the nurses and servants who had not had Small-pox, on their coming to live at the Small-pox Hospital, and not one of them has contracted Small-pox during their stay there. Re-vaccination has been extensively practised for some years past, at stated intervals, and seemingly with good effect, on the troops of some of the foreign armies, more especially and perseveringly on the armies of Germany. Combining all these circumstances together, we therefore feel perfectly justified in recommending re-vaccination for extensive adoption, after adult age, in England; especially to persons who appear, from their cicatrices, to have been but indifferently vaccinated in infancy.

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[1] See Med.-Chir. Trans. vol. xxxvi. pp. 377, 380.

For a very masterly summary of the effect produced by vaccination on the inhabitants of different parts of the world, and a thorough investigation into the whole subject of vaccination and Small-pox, at home and abroad, we earnestly recommend to all persons interested in vaccination, the perusal of the Blue Book, by John Simon, Esq., Medical Officer of the Privy Council, entitled "Papers relating to the History and Practice of Vaccination. 1857." It is to be had of Messrs. Longman and Co.

Epidemic Diffusion of Small-pox.—Epidemics of Small-pox begin generally, in London, in the autumnal period of the year. The moist weather of the autumn seems to be favourable to the spread of the disease. When, however, it has once been set going, it continues often throughout all the seasons of the year, quite uninfluenced either by the frosts of winter, or the heat of summer. A larger number of patients have, on several occasions, during epidemic outbreaks of the disease, been admitted into the Small-pox Hospital during the month of May than in any other month of the year. The present epidemic of Small-pox has lasted an unusually long time; it has now (September 1865) been going on continuously for three years. From 1796 to 1825 there was not any epidemic of Small-pox in London. After that came the epidemic of 1838. Since then epidemics have followed in rapid succession. There was one in the winter of 1840-1. Small-pox was epidemic again in 1844-5, in 1848, in 1851-2, in 1854-5-6, in 1859-60, and in 1863-4-5. The epidemic of 1844 began in May; all the rest had their commencement in the autumnal period of the year. We are quite unable to explain why the disease becomes at times epidemic, then culminates, and then declines; nothing has yet occurred to lead us to the solution of this question. The decline would not seem to be for want of subjects to act on, as of late years fresh epidemics have commenced two or three years only after the preceding epidemics had ceased, and the disease has attacked numerous persons of adult age who were residing in the same place when the disease was raging there but a short time before, but who missed taking it then. A probable explanation of this would be that during the epidemic that had just before passed, these persons were not so susceptible to the influence of the variolous poison, although they became so a short time afterwards; this would seem to be the most obvious explanation of the occurrence, whether it is, or is not, the true one.

INOCULATION FOR SMALL-POX.

It is a remarkable fact in the history of medicine and the treatment of disease, that the inventor of variolous inoculation should be unknown: even the place where inoculation originated is unknown. What we do know for certain is that inoculation was practised at Constantinople in 1700. Dr. Gregory[1] writes—"About the year 1703, rumours of the great success of this operation attracted the attention of Dr. Timoni, a Greek physician, who had studied and graduated at Oxford. He subsequently settled in Constantinople, and being convinced of the importance of the discovery, wrote an account of it, in 1713, to his English correspondent, Dr. Woodward, which, in the following year, was published in the Philosophical Transactions. In 1715, Dr. Pylarini, the Venetian consul at Smyrna, having also learnt the success of this Turkish practice, published an account of it at Venice. A notice of this work appeared in the Philosophical Transactions for 1716, and these favourable accounts were fully corroborated by the reports of Mr. Kennedy (an English surgeon, who had travelled in Turkey) in his Essay on External Remedies, published in London in 1715.

"No notice, however, was taken of these important facts by any English physician, and the idea of transplanting or engrafting Small-pox (as the process was called) was well-nigh forgotten in London, when the celebrated Letter of Lady Mary Wortley Montague appeared, which described the practice in so lively a manner as to attract public attention.[2] 'The Small-pox,' she writes, 'so general and so fatal amongst us, is here entirely harmless by the invention of engrafting, which is the term they give it. There is a set of old women who make it their business to perform the operation. Every year thousands undergo it, and the French Ambassador observes pleasantly that they take the Small-pox here by way of diversion, as they take the waters in other countries. There is no example of any one that has died of it, and you may believe I am well satisfied of the safety of the experiment, since I intend to try it on my dear little son. I am patriot enough to take pains to bring this careful invention into fashion in England.'

"She kept her word, and to the spirit and enterprise of this lady the introduction of inoculation into this country is altogether due; her own daughter was reserved to be the first example of inoculation in England. This event occurred in 1721, and its success was complete."

Through the advice of Drs. Timoni and Pylarini, inoculation was begun in America in 1721, by Dr. Boylston; 244 persons were inoculated, and six died. About the same time, in England, a son of Lord Sunderland, and the butler of Lord Bathurst, both died of inoculation. This brought the practice into discredit, and it was not until the middle of the last century that it was revived. In 1746, the Small-pox and Inoculation Hospital, London, was founded, that the poor might partake of the benefits of inoculation, which had hitherto been confined in a great measure to the rich. Inoculation was continued there until 1822. Dr. Gregory went carefully over the records of the hospital for this period, and found that only three in a thousand died of inoculation. The inoculated disease was usually very mild, but not invariably so. The great objection to it was, that it spread Small-pox just as the natural disease did. It could be set going anywhere by sending in a letter a bit of cotton-thread dipped in variolous lymph for the purpose of inoculation; so that, although the practice was of great advantage to individuals, it was very destructive to the public at large, and the general mortality from Small-pox was thereby greatly increased.

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[1] Cyclop. of Pract. Med. vol. iii. p. 748.

[2] Vol. ii. Letter xxxi. dated April 1, 1717.

About the time the hospital was founded, two brothers, Robert and Daniel Sutton, one practising at Bury St. Edmunds, in Suffolk, the other at Ingatestone, in Essex, by carrying out more fully than had been done before, the practice of treating Small-pox suggested by Sydenham, improved amazingly the mode of managing those under inoculation. Their practice was adopted by Baron Dimsdale, who obtained great celebrity as an inoculator. It consisted in giving purgative medicine, spare diet, and exposing the patient freely to cold air in the day, and making him sleep in a large airy apartment at night.

"*Phenomena of Inoculation.*—On the second day after the operation, if the part be viewed with a lens, there appears an orange-coloured stain about the incision, and the surrounding skin seems contracted. On the following day a minute papular elevation of the skin is perceptible, which, on the fourth day, is transformed into a vesicle with a depressed centre. The patient perceives an itching in the part. On the sixth day, some pain and stiffness are felt in the axilla, proving the absorption of the virus into the general mass of blood. Occasionally on the seventh, but oftener on the eighth day, rigors occur, accompanied sometimes with faintishness, sometimes with pain of the back, headache, or vomiting. The patient complains of a disagreeable taste in the mouth, and the breath is offensive, soon after which the eruption shows itself."[1]

In 1840, an Act of Parliament was passed, rendering variolous inoculation unlawful in England; the penalty for infringing this law is a month's imprisonment.

Still, however, under certain unfortunate circumstances, inoculation might be justifiably had recourse to. First, for instance, when Small-pox breaks out on board ship, and there is not any vaccine lymph at hand, or a probability of any being soon obtained, and persons are present who have neither been vaccinated, nor have had Small-pox. Secondly, when Small-pox occurs in our colonial possessions, and several months must elapse before vaccine lymph can be procured from England, or elsewhere. Under these circumstances, on board ship where Small-pox exists, or in houses in the colonies where unvaccinated persons are unavoidably compelled to remain in close proximity to Small-pox, inoculation of them would be perfectly justifiable. Then it will have to be considered and determined, as so many persons now take Small-pox after vaccination, and the badly-vaccinated suffer so severely and fatally from Small-pox, whether the vaccinated who have been badly vaccinated should have their vaccination tested by inoculation. It may be pretty well known, by attending carefully to the previous statements in this article, whether persons have been well vaccinated or not. If they have four or five good vaccine cicatrices, readily discernible, they will most likely have Small-pox in a mild form if they take it, and need not be inoculated. If, on the contrary, they have only one cicatrix that can but just be seen, or no cicatrix at all, they will probably have Small-pox, if they take it, in a severe form. Rather than run the risk of contracting a dangerous attack of Small-pox by inhalation, it would, perhaps, be wiser that they should have their vaccination tested by inoculation. It is no doubt a choice of evils, but the evil is likely to be much less of having Small-pox by inoculation than of having it by inhalation.

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[1] Gregory, op. cit. p. 750.

In ships, or in houses in the colonies, then, where there is Small-pox, and where no vaccine lymph can be obtained to vaccinate those who have not been vaccinated or to revaccinate those who have been badly vaccinated; and where the inmates cannot get away, no further harm would be done to the public by inoculating those who are presumably unsafe, and unavoidably obliged to be resident in such ships or houses. The greatest objection, as before stated, to the practice of inoculation, was from its spreading Small-pox, by infection, to other persons; but this objection would not be valid under the conditions above specified—these conditions and limitations being observed, inoculation might still fairly be considered to be admissible and justifiable, rather than to allow unvaccinated or badly-vaccinated persons to take Small-pox in the natural way.

Whenever, after carefully weighing all the circumstances of difficulty and danger, it is determined to inoculate, the variolous lymph for inoculation should be taken when *limpid*, and on the *fifth* or *sixth* day of eruption, and when practicable it should be chosen from a mild form of disease, and inserted into the arm in but one place, the object to be aimed at being to give Small-pox in the mildest possible way.

VACCINATION.

By Edward Cator Seaton, M.D.

Vaccination, or Vaccine Inoculation, is the process by which a peculiar specific disease—Vaccinia or the Cow-pox (from the Latin, *vacca*, a cow)—is introduced into the human system with the view of protecting it against an attack of small-pox.

The Cow-pox never originates spontaneously in man, but (as its name implies) is a disease natural to, and it is not unfrequently seen in, the cow; particularly, and indeed as a disease of spontaneous origin almost exclusively, in the milch cow. As observed in that animal it is a vesicular affection, occurring either casually or as an epizootic, the vesicles manifesting themselves chiefly or entirely on the teats and udder. It has a very precise and definite course. About four days from the probable period of invasion, without any or with scarcely any apparent general indisposition, small, red, rather tender papules appear near the udder and on the body of the teats. These become developed into vesicles, the most characteristic of which are soon seen to have an elevated margin, a central cup-like depression, and by the eighth or ninth day generally a pale rose or light damask areola, not more than a line or two in width, about the base. In two or three days more the disease has reached its acme; the areola has extended to four or five lines, with circumscribed induration of adjacent skin and subjacent cellular tissue: the vesicles, if they have not already burst, are turgid with lymph which, before perfectly clear, is now becoming opaque. By the twelfth day the lymph is more decidedly turbid and a process of desiccation and incrustation has begun, which in five or six days more is complete. The crusts so formed separate spontaneously at from the twentieth to the twenty-fourth day from invasion, leaving behind them permanent slightly-depressed cicatrices. From traction in the process of milking, the vesicles when fully formed very generally burst, and the lymph which exudes from them is frequently found to produce sores of a definite and similar character on the hands of the milkers, who, in their turn, transfer the lymph to, and become the means of infecting, other animals in the dairy.

A popular notion which existed in the county of Gloucester, as in some other dairy districts, that milkers who had thus been infected were afterwards insusceptible of small-pox, attracted about a century ago (in 1768) the attention of Edward Jenner, at that time apprentice to a surgeon at Sodbury, near Bristol. The impression then made on his mind was never effaced. At a very early period of his medical career he set himself to investigate the truth of the popular belief; and, in the course of inquiries and reflections which satisfied him on this point, his genius conceived the idea that it might be possible to propagate the Cow-pox at will in man, first by directly inoculating it from the cow and then from one human subject to another, and that therewith protection against small-pox might be imparted in perpetuity. Many years, however, elapsed before he was able to put this idea to experimental proof. But when at length he communicated to the world (in 1798), in his Inquiry into the Causes and Effects of the Variolæ Vaccinæ—"that masterpiece of medical induction," as it has justly been called[1]—his great discovery of Vaccination, he had already established on a sufficient basis of observation and experiment the following among other points relative to the Cow-pox: (1) That this disease casually communicated to man has the power of rendering him insusceptible of small-pox; (2) that the specific Cow-pox alone, and not other eruptions affecting the cow, which might be confounded with it, had this protective power; (3) that the Cow-pox might be communicated at will from the cow to man by the hand of the surgeon, whenever the requisite opportunity existed; and (4) that the Cow-pox, once ingrafted on the human subject, might be continued from individual to individual by successive transmissions, conferring on each the same immunity from small-pox as was enjoyed by the one first infected direct from the cow.

It is in this transmission of Vaccinia from one human subject to another that the practical usefulness of Jenner's discovery lies: and since the process was first made known, all ordinary vaccinations have been thus carried on. Indeed, although at convenient opportunities, or on grounds which will be hereafter stated, lymph has at various times since Jenner's day been obtained direct from the cow, and new stocks, as it were, thus introduced into practice, we may safely say that the great bulk of the vaccinations of the present day, in this country at least, are performed with lymph transmitted from the early direct vaccinations of Jenner himself.

In endeavouring to give a brief account of the present state of our knowledge in regard to Vaccination, I shall (1) describe the phenomena of Cow-pox as observed in the human subject; (2) describe the mode or modes of inducing these phenomena, or of vaccinating; (3) review the evidence we have of the extent and degree of protection against small-pox which Vaccination affords; (4) state the pathological relations of variola and vaccinia; and (5) inquire whether Vaccination, in protecting mankind against one disease, is, or may be, the means of introducing others—whether, in short, the good that it is known to effect is counterbalanced by any, and what, degree of possible harm.

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[1] Simon, Letter prefixed to Papers relating to the History and Practice of Vaccination, p. xii.

I. PHENOMENA OF COW-POX IN THE HUMAN SUBJECT.—The symptoms which Cow-pox manifests in the human subject resemble very closely those already described as observed in the cow. If some vaccine lymph be taken on the point of a lancet, and inserted by puncture on the arm of an infant who has not before been vaccinated, no particular local effect is noticeable for the first two days; but, if the Vaccination be about to succeed, by the end of the second or by the third day a slight papular elevation is perceptible, which, by the fifth or sixth day, has become a distinct vesicle of a bluish-white colour with a raised edge, and a peculiar, central, cup-like depression. By the eighth day (the day week from the insertion of the lymph) this vesicle has attained its highest perfection, is plump, round, and more decidedly pearl-coloured; the elevation of its margin, and the depression of its centre, are more marked. At this date, or sometimes a few hours earlier, a ring of inflammation, termed the areola, begins to form about its base, and, for the next two days, continues to spread. It is circular, and, when fully developed, has a diameter of from one to three inches, and is often attended with considerable hardness and swelling of the subjacent connective tissue. The establishment of the areola demands always the attention of the practitioner and student, as the anatomical evidence that the Cow-pox has produced its specific effect on the constitution. Other proofs of the constitutional influence of the Vaccination are, at this period of its course, generally afforded in the child's restlessness and heat of skin, with (frequently) derangement of the stomach and bowels, and with (sometimes) swelling of the axillary glands. But these general symptoms, though seldom altogether absent, are often exceedingly slight; nor, provided the areola be properly formed, are they to be looked upon as indispensable for the protective effect of Vaccination. After the tenth day the areola begins to fade, the vesicle begins to dry in the centre, the lymph remaining in it becomes opaque and concretes, and, by the fourteenth or fifteenth day, a hard brown scab is formed, which contracts, dries, blackens, and, from the twentieth to the twenty-fifth day, falls off, leaving a cicatrix commonly permanent, and which in character is circular, somewhat depressed, foveated or indented with minute pits, and sometimes radiated.

Occasionally certain constitutional symptoms beyond those already described are observed. In young children of full habit, especially in hot weather, about the ninth or tenth day, when the areola is at its height, an eruption of roseola will sometimes take place, chiefly on the extremities; sometimes the eruption has a papular form (vaccine lichen), and sometimes it is vesicular. These eruptions are generally very transitory; their ordinary duration does not extend beyond a week; and they very seldom indeed last beyond the falling of the scab.

When the mode of inserting or applying the lymph has not been by single puncture, or by such abrasion of the skin as would raise a single vesicle, but by two, three, or more punctures close-set together, or by scarifications or abrasions over some extent of surface in the manner that will hereafter be described, so that two, three, or more vesicles are developed in close proximity, these usually coalesce, and a vesicle results, the compound character of which is very obvious: or there may be a crop of coalescent vesicles, each having its distinct head with characteristic depression. These compound vesicles and crops are round, oval, or of irregular outline, according to the manner in which they have been induced, and the shape of the resulting cicatrices varies accordingly.

The clear, smooth, supple, delicate skin of the infant is peculiarly adapted to manifesting in perfection the local characters of the vaccine disease. In elder children, and much more in adults, though the phenomena of primary vaccination are essentially the same as in the infant, the vesicle is often wanting in that plumpness, sharp definition of edge, and beautiful lustre which the experienced vaccinator delights to see. And, in the adult, the course of the disease is frequently somewhat retarded, the areola is apt to be more diffuse, and swelling of the axillary glands is more frequently observed.

When lymph is employed that is derived directly or very recently (as within three or four removes) from the cow, the course of the disease is generally retarded at various stages. Papulation is sometimes deferred till the seventh, eighth, ninth, or even the tenth day, and the areola is not complete till from the eleventh to the fourteenth or even the sixteenth day. The areola, when at its height, is more indurated than is observed in Vaccination with ordinary humanized lymph, and is said to decline and revive, continuing to exhibit a brick-red or purplish hue while the hardness remains.[1] The papular and vesicular eruptions, which have been above referred to as occasionally attending Vaccination at this stage of its course, are more frequently seen. The vesicles themselves are commonly not more developed than those produced by ordinary lymph. Desiccation is generally prolonged, and the crust is often retained till the fourth or fifth week.

Such are the ordinary phenomena induced by primary vaccination in the human subject. Their course, however, is sometimes modified; and may be (*a*) simply retarded, (*b*) simply accelerated, or (*c*) altogether irregular and spurious. (*a*) *Retarded Cow-pox.*—The most simple and frequently seen form of retardation is a mere delay for a day or two in the course of the vesicle; by the eighth day it has not more size or development than ordinarily is met with on the sixth, and the areola does not form till the tenth day, or even later. This amount of retardation is probably more frequently met with in cold than in hot weather, especially if the cold be accompanied with a dry easterly wind. Sometimes the delay is much longer; at the end of a week from the Vaccination, when the child is brought for inspection, so little of result is seen that it is a question whether the Vaccination has not altogether failed; some fresh lymph is inserted on another spot, and, as the vesicle of the new Vaccination rises, that of the first Vaccination is seen also to develop itself, and the two run their course at the same time. Bousquet refers to a case (not in his own practice) in which this revival of a dormant Vaccination took place, it was said, at the end of three weeks.[1] I have never seen any case in which the vesicle rose after such a delay as this; but I have seen cases in which, without any recourse to a second Vaccination, vesicles have risen a week after the usual period, and when success had ceased to be looked for. Retarded Cow-pox is much more frequently seen when Vaccination is performed with dry lymph than when it is done direct from the arm; no doubt because, in the former case, the lymph, when deposited on the cutis, is often in an undissolved, or imperfectly dissolved, state. Mere retardation of phenomena, if these phenomena be regular in their character, does not in any way interfere with the protective value of the Vaccination. Retardation of Vaccination at various stages of its course is often independent of any obvious cause; but sometimes it can be traced to certain conditions of the system, as the incubation of other diseases. If, for instance, vaccine lymph has unwittingly been inserted in a child who happened to be incubating measles or scarlatina, and the Vaccination has succeeded, the vesicles will usually, when the rubeolar or scarlatinal symptoms set in, be arrested in their course, and no areola will be formed till the measles or scarlatina have subsided.[2] Again, when Vaccination has been performed with effect on a child who is incubating small-pox, the vaccine vesicles usually proceed in the ordinary way till the small-pox manifests itself, and, even after this, may undergo some further local development, the vaccinia and variola apparently going on together; but unless, before the small-pox appeared, the Vaccination had already reached the stage of areola, its progress to that stage will be arrested, in which case it will have no effect in modifying the small-pox. (*b*) *Accelerated Cow-pox* is to be regarded with much more suspicion than retarded Cow-pox, spurious vaccination having generally an accelerated course. There may be, however, simple acceleration; a course some twelve or twenty-four hours in advance of the usual course, the vesicle on the eighth day being in the state in which it is usually seen on the ninth, the areola nevertheless being regular, and the crust being subsequently duly formed. In such case the value of the Vaccination is not impaired. (*c*) *Irregular and Spurious Cow-pox.*—Vaccination sometimes runs an entirely irregular course, the varieties of irregularity being considerable. In one form, the most frequent, the course resembles that of a revaccination (as will hereafter be described) instead of that of a primary Vaccination; the vesicle begins with itching and irritation; it is acuminated or conoidal, instead of being flat and with central depression, and it contains straw-coloured or opaque fluid, instead of clear lymph; it has an early or irregular areola, which is at its height by the fifth or sixth day, and when seen on the eighth day is far on the decline; there is then a small scab on the surface which usually drops off by the tenth day. Or the local effect may have been even less than this, and on the eighth day there may be nothing remaining but a very thin scab or scale just about to detach itself, and which at the lightest touch falls off. In other cases, the vesicles, when seen on the eighth day, are found to have burst, and to present an irregular pustuloid or scabby appearance, or to be so many open sores. It would be difficult, and is quite unnecessary, to give a verbal description of each form of irregularity that may be observed; *the one important practical fact being that a Vaccination presenting any deviation from the perfect character of the vesicle, and the regular development of the areola, is not to be relied on as protective against small-pox.* A spurious and irregular course of the vaccine vesicle, no doubt, sometimes occurs without assignable cause, and in the most experienced hands, but it is far more usually due either to the use of lymph not well chosen by the vaccinator, or to something amiss in the state of the child vaccinated. Thus, it may be seen in various forms on several children vaccinated from the same source; or it may occur in children who are vaccinated from undeniably good sources, but who themselves, at the time, are suffering from intertrigo or other affections. A spoilt and broken appearance of vesicle, which is frequently on the eighth day ascribed to rubbing or mechanical irritation, is often really an irregularity arising from one or other of these causes; and the best proof how much such irregularities depend on the care and skill of vaccinators, is the frequency with which they are seen in the hands of some vaccinators, and their extreme rarity in the hands of others. Parents, however, should always be directed to be very careful to keep the vesicles uninjured in their progress, and to avoid the premature removal of the crust.

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[1] Ceely, Observations on the Variolæ Vaccinæ in Trans. of Prov. Med. and Surg. Assoc. vol. viii. p. 346.

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[1] Nouveau Traité de la Vaccine, p. 176.

[2] Jenner notices this in one of his early tracts, Continuation of Inquiry, p. 31.

The local and general symptoms of Vaccination seldom call for any treatment. Occasionally, however, they run an active course; the arm becomes inflamed; erythema, or sometimes true erysipelas, develops itself; the vaccine vesicle degenerates into a purulent ulcer, or sometimes into a sloughing sore, leaving a cicatrix which has none of the characteristic pittings, but is simply a puckering, or a flat, smooth, shining scar. The treatment of such cases would be the treatment of erysipelas or of inflammation from any other cause. It is needless to say that degeneration of the vesicle thus taking place deprives the Vaccination of all protective power.

Phenomena of Revaccination.—In the majority of persons the regular phenomena of Vaccination, such as we have described them, can only be produced once in the lifetime; any subsequent introduction of lymph either failing to produce any local effect whatever, or (much more commonly) producing a modified effect, resembling one of the forms of spurious Vaccination. The absence of effect is relatively most common in the child, the spurious effect most common in the adolescent and adult. This spurious effect consists either in a papule, or (more often) in an acuminated vesicle, with a hard and irregular areola. The symptoms begin early, reach their height by the fifth or sixth day, and then decline. The scab, small and imperfect, forms generally on the eighth day, and soon falls. There is usually much itching, and often considerable constitutional irritation. Severe constitutional symptoms are, out of all proportion, more frequent in revaccination than in primary Vaccination; and in very exceptional cases the vaccine lymph may act as an animal poison, giving rise to phlegmonous erysipelas: some still rarer cases have occurred of pyæmia, terminating fatally. In a certain proportion of cases, the results of revaccination are the same as those of primary Vaccination, the vesicle, in shape and character, being in no degree distinguishable; in such cases the areola is sometimes small and transitory, and the scab on falling leaves a small inferior cicatrix; at other times the areola is perfect, and a good cicatrix is left. Normal vesicles resulting from revaccination are much more frequent in adults than in children, but I have seen them in the arms of children a few years old, who had excellent marks of their first Vaccination.

II. METHOD OF VACCINATING.—For the proper and successful conduct of Vaccination, special attention must be given to the state of health of the child to be vaccinated, to the selection of the lymph to be used in the Vaccination, and to the thorough insertion of it.

1. Except for pressing reasons, children should only be vaccinated when they are in good health. Especially must they be free from any acute disease, from diarrhœa, or from any chronic disease known to interfere with the regular course of the vaccine vesicle. Of these diseases, the chief are herpes, eczema, and intertrigo. The latter affection, especially, so often modifies, or even completely spoils the course of Vaccination, that it is of great consequence, before proceeding to vaccinate a child, to see that there is no chafing behind the ears, in the folds of the neck, or in the groins. But cutaneous eruptions of all kinds are a contra-indication, and, as a rule, great attention should be given to having the skin perfectly clear, and free from them, before inserting vaccine lymph. Of course this rule is liable to be modified by circumstances; and especially when there is any immediate risk of small-pox, it becomes imperative to perform Vaccination notwithstanding these contra-indications.

Health permitting, all children should be vaccinated in very early infancy. Inasmuch as one-fourth of the deaths from small-pox in England occurs in children under the age of one year,[1] it is obvious that delay is attended with extreme risk; and this risk, it need scarcely be said, is greatest in large towns. Plump and healthy children, living in large towns, should be vaccinated when a month or six weeks old;[2] in more delicate children, the Vaccination might be postponed till they are two or three months old; but all, except those whose state of health positively contra-indicates Vaccination, should be vaccinated by the age of three months. This early period of life is also particularly suitable for Vaccination, as being usually free from the disturbing influence of teething. Circumstances connected with lymph-supply render a longer delay unavoidable in many instances in small towns and country districts; and, when small-pox is not present in the localities, such delay, if not too long continued, is not of material moment. But even children living in these districts should always be vaccinated within a few months, six or seven at the utmost, from birth.

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[1] Of 42,277 deaths from small-pox which occurred in England in the ten years 1856–65, 10,223 were in children under one year of age.

[2] If the parents of a child, apparently healthy, were known to have any taint of syphilis, it would, in the interests of Vaccination generally, be a matter of prudence to postpone the Vaccination, unless small-pox were imminent, till the age was passed

Under circumstances, however, of direct exposure to small-pox, it should be well understood that no age is too early for Vaccination; infants have repeatedly been vaccinated immediately after birth, and thereby saved. In all cases of much risk, it is of the utmost importance to avoid any delay. The loss of a day may be the sacrifice of a life. The Vaccination may possibly be in time to prevent small-pox altogether; but even supposing the variolous infection to have entered the system before the Vaccination has been performed, still if *the Vaccination be only got to the stage of areola* before the small-pox manifests itself, it will exert its modifying power, and the child will be saved.[1]

2. The lymph to be used in vaccinating should be taken from healthy subjects, and from thoroughly characteristic vesicles. It *may* be taken as soon as the vesicle will yield it, when, though it can only be got in small quantity, it is very effective. It is usually taken, and for the ordinary purposes of Vaccination is best taken, when the vesicle is fully formed, but either before the areola appears, or within a very few hours of its commencement. This, in regular cases, is on the day week from the Vaccination; the lymph is then easily obtained in sufficient quantity. It is a very great mistake, but one which is often made, to take it at a later period of the vesicle than this, and when the areola is fully complete. No doubt it flows then more freely, and may be got in greater abundance; no doubt also (as alleged by those adopting this practice) it very often takes; but it does not do *this with anything like the same certainty*—a point of great consequence; and it is also more likely to lead to erysipelatous and spurious results. Prime lymph has always a certain degree of viscidity, and a thin serous lymph, even from a vesicle which is not advanced, is to be avoided. Babies are much better lymph-givers than elder children, or adults.

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within which hereditary syphilis mostly manifests itself, *i. e.* till the completion of the third month from birth. For if any syphilitic symptoms were to appear after the Vaccination, they would most likely be ascribed not to their hereditary cause but to the Vaccination.

[1] The incubative period of small-pox being twelve days, and the time requisite to carry Vaccination to the stage of areola being only nine days, it is manifest that even a person who has actually imbibed the infection of small-pox may, by Vaccination within the first three days after the reception of the infection, obtain the modifying benefits of Vaccination. A day more, and the Vaccination will be too late: whatever local effect it may produce, the small-pox will not be modified by it. (See the article Small-pox; see also the subject more fully treated in my "Handbook of Vaccination," pp. 102–106.)

Children of dark complexion, not too florid, with a thick, smooth, clear skin, yield the finest and most effective lymph.

3. A child and a vesicle fit for the purpose having been selected, the vaccinator in order to collect the lymph proceeds to open the vesicle by a number of minute punctures, which must be made on its surface and not round the base. The object in making many punctures is to open the various cells of the vesicle, and the reason for making these on the surface, and not round the base, is to obtain the lymph free from any admixture of blood. If by accident any blood be drawn, this must be allowed to coagulate, and then be carefully removed before taking the lymph; for it is a cardinal rule, never to be deviated from, that the inoculation must be with vaccine lymph, *and with lymph only.* When the cells of the vesicle are freely opened, the lymph soon exudes, and lies on the surface. The lancet or point to be charged is then dipped into it; or if a capillary tube is to be charged, the end of the tube is inserted in the liquid as it lies. On no account must there be any pressure or squeezing of the vesicle with the lancet to make the lymph exude: and when lymph ceases spontaneously to stand on the surface of a vesicle, that vesicle must be considered no longer usable for lymph-supply. Very generally, however, when the lymph which has first exuded has been taken, and the surface of the vesicle left apparently dry, if the operator wait a minute or two, he will find there has been a fresh exudation of good usable lymph; and when he does not find this, he may often induce it in a way quite unobjectionable, by wiping very gently the surface of the vesicle with a soft wet linen cloth, thereby removing or dissolving the inspissated lymph which clogs the punctures. Vesicles of perfect character, and of the same size and appearance, differ very much in their yield of lymph; ordinarily, from a vesicle of such size as is produced by a single deep puncture, enough lymph may be got for the direct Vaccination or from four to six children, or for charging (*i.e.* for well charging, dipping once and again) six to eight ivory points. Some vesicles yield much more, but the caution already given against a thin, serous, too-readily-flowing lymph must be borne well in mind. When vesicles are compound, their yield of lymph is of course proportionally increased.

Lymph should in every instance (where practicable) be inserted direct from arm to arm. All processes for preserving and conveying lymph (valuable, and indeed invaluable, as they are for their own proper purposes) render in the long run the taking of the Vaccination a matter of inferior certainty:[1] and it is only in cases of necessity that Vaccination should be performed with lymph stored or conveyed. But a caution must be here interposed. The superior relative advantages of arm-to-arm Vaccination are so well known, that practitioners whose vaccinations are few, and whose opportunities of lymph-selection are therefore limited, will often be induced to take their lymph from second-rate vesicles, rather than lose the opportunity of vaccinating direct from an arm. This, which I know to be the source of much current inferior Vaccination, is a course which should never be adopted. The operator, *unless he has some preserved lymph that he can rely on, and unless he can rely on his own hand to make preserved lymph take*, should put off the Vaccination till he has the opportunity of doing it from a thoroughly satisfactory vesicle.

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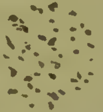
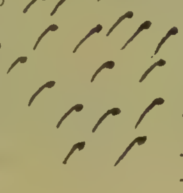
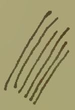
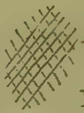
[1] An account of the various methods of storing lymph and their relative advantages is given in my "Handbook of Vaccination," chap. viii.

Various methods may be employed for inserting lymph—the essential part of all of them being either to introduce the lymph into the substance of the cutis, or to bring it well in contact with its absorbing surface. (*a*) One of the most commonly used is that by puncture. When it is intended to operate in this way, the arm of the child to be vaccinated should be grasped by the left hand of the vaccinator, so as to put the skin on the stretch, and a very sharp, perfectly clean lancet, well charged with the lymph selected, should be introduced by valvular puncture from above downwards, so that the lymph may gravitate into the wound. The lancet should not be held level with the skin, but at an angle of 45°, or thereabouts, and made *to enter the cutis.* If the lymph be thus well put in, it is retained by the valvular character of the puncture and the elasticity of the skin; and any fear that the bleeding which ensues will cause the Vaccination to fail is quite chimerical. A minute and superficial puncture, on the other hand, does not unfrequently fail. In vaccinating by puncture, *not less than* five should be made, and they should be at a distance of half an inch from each other. Five or more punctures at this distance from each other can very well be made on one arm, or, if the operator prefer it, three or more may be made on each arm. In the manipulations by which Vaccination is effected by puncture, it is a very good plan to make each puncture a double one; thus, //. A finer and larger, often oval, compound vesicle is thereby raised. For Vaccination by puncture no instrument is needed but a common lancet, very sharp: and many of the instruments which have been specially devised, are not, in my opinion, nearly so good. (*b*) A modification of the plan of vaccinating by puncture is that of multiple superficial punctures, or tattooing—a number of minute superficial punctures being made with the point of the lancet, thus, and the lymph then spread over with the flat part of the lancet. The number of spots over which this tattooing should take place will depend, of course, on the extent of surface operated on at each spot; but in order that a local effect may be produced equal to that of five ordinary vesicles, tattooing over such a surface as is above depicted should be repeated on at least three spots. (*c*) Another modification of puncture, common in some of the northern districts of England, is that of first spreading the lymph on the arm of the child to be vaccinated, and then ripping up the cuticle with the point of the lancet over a surface equal to a sixpenny-piece, or more, with, frequently, a second plastering of lymph afterwards; crops of vesicles are thus raised, close set together, and nearly always confluent, each vesicle, however, having its distinct depressed head. I have counted as

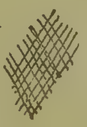




many as eighteen or twenty such on one base; but the vesicles by the pressure they exercise on each other do not attain the size and development of separate vesicles. It is usual to make two crops, such as I have described, and this must be regarded as sufficient for full protection. (*d*) Vaccination is often performed, not by puncture, but by scratch; three or four longitudinal or transverse scratches, each about three-quarters of an inch long, are made at distances half an inch to an inch apart from each other, with the point of a lancet, or with a thick needle, and the lymph is rubbed on. In the course of each scratch two or three separate vesicles will arise, or more frequently, one oblong compound vesicle will be produced. (*e*) Another and very excellent plan of scarification is adopted by many vaccinators, which consists in abrading the cuticle by a number of fine parallel scratches, thus,  or by further cross-scratch thus,  Abrasion should only be carried so far as to make it certain by the appearance of blood oozing, that the cutis is reached; any such oozing is then rapidly wiped away with the finger, and the lymph plastered on. When the abrasions are made to cover such a space as is above indicated, it is sufficient to make two on each arm, or three on one arm, about three-quarters of an inch or an inch from each other—but when, as by some practitioners, the abrasions are made over smaller surface, producing vesicles which are not more than equal in size to those of good puncture, there should be at least five such abrasions. Vaccination by scarification cannot be better done than with a common lancet: some practitioners, however, prefer the use of a scarifier or rake (invented by Dr. G. Weir), which consists of three or four needle points, inserted in a handle of ivory. The skin being held very tense, this rake is drawn lightly across in one direction, and then, if the operator pleases, crossed in another, the lymph being then plastered over.

In cases in which Vaccination has to be performed with preserved lymph, if the lymph has been preserved liquid, as in Husband's tubes, no other directions are requisite for the performance of the operation than those which have now been given. But if the lymph has been preserved by drying, as on points or glasses, it will be necessary that it should be revived or brought back to the liquid state in order that it may be taken up by the system. This is done by the use of a very minute quantity of water; if the lymph to be revived has been preserved on glasses, a very small drop of water is taken on the point of the lancet and well rubbed in with the lymph, which is then left for some minutes to soften; or, if the lymph has been kept on points, these should be very lightly dipped in water and then placed for a few minutes on the edge of a book. Much care is required in thus reviving lymph: on the one hand there must be moisture enough to bring the lymph back to the state in which it was when taken from the vesicle; on the other, it is important that the lymph be not too much diluted. For puncture-vaccination the revived lymph is either taken on the lancet-point and inserted as in a Vaccination direct from the arm, or, if the mode of preserving has been by points, a puncture is first made with a clean uncharged lancet and the ivory point is then itself inserted into this puncture and kept in position by the thumb for a short time, care being taken to press the thumb well down as the point is being withdrawn, and thereby insure that the lymph on it is well wiped off and left in the wound. In Vaccination by scarification or abrasion, the flat of the glass, or the flat of the point, may be rubbed over the scarified or abraded surface.

Of the different modes of vaccinating which I have described, and of numerous modifications of them which are met with in practice, is there any which can be more thoroughly relied on than another for infecting the system? This is a question very frequently put to me, and my reply always is that a person properly taught *ought* to be able to infect thoroughly by either of them; and that any one who has so learnt, and so habitually practises, either plan as to be able to rely on securing the results he aims at, should not—so much depends on habit—change it for any other. By every one of the processes I have described, I have seen the most perfect results produced by experienced vaccinators: certainly by no process more completely than by the one I first described—that of simple puncture. But unusual opportunities of observing Vaccination as generally practised have shown me that many, if not most, practitioners do not—for want of original instruction, and of attention to various small but essential points—by whatever process they adopt, succeed as they ought to do; and judging by the results in the cicatrices which their vaccinations leave, that, as a rule, those who vaccinate by simple puncture are by far less successful than those who vaccinate in either of the other ways described;[1] further, that the best marks, on the whole, as regards size, depth, and foveation, are those which result from the plan last of all described, viz.: that of cross scratch or abrasion over a sufficient surface. And this plan is, in most hands, eminently more successful than puncture when dry lymph has to be employed.



Mr. Marson states that "with good lymph, and the observance of all proper precautions, an experienced vaccinator should not fail of success in his attempts to vaccinate above once in 150 times."[2] His own habitual success is, indeed, far greater than this; but so much, at all events, he considered might fairly be expected of any good vaccinator. That this standard is not too high is clear from inquiries I have made as to the results obtained at several large stations where Vaccination is well performed and where accurate records are kept, at which I found that the average of failures to infect at the first operation did not exceed one in 170 cases. Good vaccinators expect also to raise a vesicle for every point of insertion; and, in their hands, cases in which only one or two vesicles result from four or five punctures or scarifications are as rare as total failures. But such results, it must be well understood, will not ensue unless patience and diligence be given to learn how to select lymph, and how to manipulate; nor unless practitioners, seriously considering the responsibility which rests upon them when they are proceeding to vaccinate, act with corresponding care. If they fail to infect their patients at the first, and still more if they fail at the second trial, it is very likely these will not again present themselves, and will remain unprotected. If, endeavouring to raise four or five vesicles, they raise only one or two, their patients do not get all the protection Vaccination is capable of affording. It has happened to me for many years past to see, all but daily, children growing into manhood and womanhood, who were unprotected against small-pox, not by their own or their parents' neglect of Vaccination, but by the want of skill of operators, who (often with two, three, and four operations) had failed to infect them. Mr. Marson also says, "Patients often present themselves with small-pox at the hospital, who state they have been cut five, six, or eight times, or more, for cow-pox without effect." "This," he adds, "is a great evil. It would happen but rarely in careful hands." So far, indeed, are practitioners generally from having attained the standard which all should reach, that it is no uncommon thing to hear 5 and 10 per cent. of failures spoken of as a satisfactory result, while not a few think they do well if they succeed in four operations out of five: and constantly practitioners are met with, who do not in one-half the cases they vaccinate, and many of them not in a quarter, raise all the vesicles which they have attempted to produce.[1]

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[1] Chiefly, I believe, on account of the minute and superficial character of the puncture, and the very small quantity of absorbing surface exposed to the action of the lymph. This arises in great measure from a chimerical fear that if blood be drawn the lymph will be washed away. There is a kind of Vaccination by scarification which is equally imperfect in its results, when a sort of make-believe scratch like this | is made, and a little lymph put on.

[2] Analytical Examinations of Small-pox and Vaccination, Medico-Chirurg. Trans. vol. xxxvi.

Insusceptibility to the infection of Cow-pox, even for a very limited period, is an excessively rare occurrence. For in the few cases in which good operators fail at the first operation to infect they rarely fail at a second trial, but now and then this is so, and even a third operation may not take. In such cases, if the operations have been properly performed, and lymph inserted direct from the arm, it may be assumed that there is temporary insusceptibility. Cases have been mentioned to me on good authority, but I have never met with such myself, in which this insusceptibility has remained for many years.

III. PROTECTION AFFORDED BY VACCINATION AGAINST SMALL-POX.—Persons who have once been successfully vaccinated are, as a rule, permanently protected against small-pox. A certain but indeterminate proportion of vaccinated persons will, however, be liable at some period or other of their lives, especially under epidemic influence, to take small-pox in a mild and modified form. A very much smaller proportion will be liable to take it in a severe, disfiguring, or fatal form. But the liability of any individual to take small-pox severely after Vaccination, and probably the liability to take it at all, will be inversely as the goodness and amount of the Vaccination.

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[1] *Vide* Reports of Medical Officer of Privy Council, iii. iv, v. vi. and vii.

The protection which Vaccination is capable of affording against small-pox was held by Jenner to be exactly that—neither more nor less—which an attack of small-pox, either taken naturally or induced by inoculation, was well known to confer against a subsequent attack of the same disease. Believing Cow-pox and small-pox to be identical, he considered that the system of a vaccinated person had already in fact passed through an attack of small-pox. He was well aware that this disease did in some individuals recur, and that the having passed through one attack was not in every instance a security against a future attack. He refers repeatedly in his writings to cases of this kind seen by himself and by other reliable observers, and states the claims of Vaccination thus:—"Duly and efficiently performed, it will protect the constitution from subsequent attacks of small-pox as much as that disease itself will. I never expected it would do more; and it will not, I believe, do less." It was only, however, to *efficient Vaccination*, i.e. to Vaccination which had gone through all its stages with perfect regularity and had given evidence of infecting the constitution, that he attributed this protecting power. Inefficient Vaccination, like inefficient inoculation, he knew would fail. Observations made since Jenner's day, with remarkable care and ability and on a scale which gets rid of all sources of fallacy, by Mr. Marson of the Small-pox Hospital, have conclusively established that, for *thoroughly* infecting the constitution, a certain *amount* of local affection is as necessary as a perfect *character* of vaccine vesicle. We must therefore so far extend the meaning of the words "due and efficient" performance of Vaccination, as to make it include amount as well as quality of vaccine influence: and with this extension, the experience of seventy years tends to show the correctness of Jenner's estimate.

The protective power of Vaccination, like that of natural or inoculated variola, shows itself in two ways: 1st, as regards the large majority of persons, in shielding the constitution against any future attack, however modified, of small-pox; and 2d, as regards the remainder, in generally so modifying the subsequent small-pox that that disease is, as a rule, deprived of all danger to life, and does not on recovery leave behind it those disfiguring traces which are not the least of the terrors of unmodified variola.

1. On no subject is medical testimony more unanimous than on the very large immunity from attacks of small-pox which successful Vaccination will confer.[1] While there are few unvaccinated persons

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[1] See this testimony as given on a large scale in Report of Small-pox and Vaccination Committee of Epidemiological Society, 1853; Seaton on Protective and Modifying Powers of Vaccination, 1857; and Simon, Op. cit. pp. lxxix. lxxx. The Epidemiological Society refer to answers received from above 2,000 medical practitioners in England, besides evidence collected from abroad, affirmative of the protective value of Vaccination. Simon gives the answers, to the like effect, of 540 distinguished medical men, British and foreign (out of 542 of whom inquiry was made), to a question "purposely con-

who do not at some period or another of their lives sustain an attack of variola, the vaccinated are, as a rule, entirely exempt from it. Precise data have never been collected on a sufficient scale to enable us to state with numerical accuracy what proportion of persons may still be liable to take small-pox, who have already had variola, or who have been successfully vaccinated. The proportion in both cases will vary of course with the degree of exposure to the infection, and will no doubt also be greatly dependent on the presence or absence of epidemic influence. We have, however, as regards one limited class of the population, some very precise facts. The records of the Royal Military Asylum at Chelsea show that 5,774 boys were admitted into that institution in the course of the forty-eight years ending December 1851, of whom 1,950 had on admission marks of small-pox, and 3,824 either had marks of Vaccination or were on admission vaccinated:[1] of the former 6·15 per thousand, and of the latter 7·06 per thousand, contracted small-pox subsequently during their residence in the asylum. Dr. Balfour, the able and accurate statistician to whom we are indebted for these facts, tells us also that in a long series of years antecedent to the publication of his Memoir,[2] the annual number of *cases* of small-pox in the British army had not been more than 6·6 per 10,000 men, one-fifth of the men, it was calculated, being protected by previous small-pox, and the other four-fifths by Vaccination. That portion of the troops which was serving in the United Kingdom, and was quartered chiefly in large towns (from which, to the disgrace of this country, small-pox is seldom absent), suffered in greater proportion, but still the average annual admissions among these were for a period of ten years (1837-46) only 22 out of every 10,000 men. The returns for the four years 1859-62 show on the average a considerable diminution even in this ratio.[3]

Year.	Number of Troops in United Kingdom.	Cases of Small-pox.	Deaths.	Per 10,000.	
				Cases.	Deaths.
1859	71,715	175	7	24·3	0·97
1860	85,443	140	9	16·8	1·05
1861	88,955	51	4	5·9	0·45
1862	78,173	64	4	8·1	0·51

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structed to elicit the expression of every existing doubt on the protective influence of Vaccination."

[1] This period extends back as far as 1803 : it will be noticed that one out of every three children admitted in these forty-eight years showed marks of small-pox. No better illustration of the progress Vaccination has made and of its wonderful influence in protecting against small-pox can be given, than is afforded by contrasting this proportion with the proportion we *now* find of children of our poorer classes who are marked with small-pox. When, five years ago, an examination was made among school children of this class in London, less than one in forty was found to have any traces of small-pox.

[2] In 1852 ; in Medico-Chirurg. Trans. vol. xxxv.

[3] The variation in the annual number of cases depends on the presence or absence of epidemic influence : two epidemic and two non-epidemic years are included in the table.

In civil practice, numerical inquiries have sometimes been made as to the protection enjoyed by individuals under the highest degree of exposure to small-pox—members of families in which that disease existed, who were living and sleeping at home, in the same house, even generally in the same room, and sometimes in the same bed, with the infected case. The result of such an inquiry, made by Mr. Cross, of Norwich, was that of 215 unprotected members of families so circumstanced, 200 contracted the disease and 46 died; while of 91 vaccinated, only 2 took the disease, and these both had it in its modified form. A rather more extensive inquiry was made some years later by Mr. Marshall, of Chelsea,[1] into the facts connected with 757 individuals in infected families. He found that of 231 who had been protected by Vaccination, 27 had contracted small-pox during an epidemic that had just then prevailed; that of the unprotected every one had been attacked except 7; and that 14 cases had occurred in persons who had previously had variola. The only inquiry of this kind, so far as I know, in which regard has been had, not merely to the fact of Vaccination, but to its amount and quality, has been the one made some years ago by the Epidemiological Society, as to the extent to which medical men—who, from their profession, would be unusually exposed to small-pox—had suffered from that disease after Vaccination or after previous variola. The results of this inquiry were, that of 347 who had been vaccinated, 44, or 12·6 per cent. had subsequently contracted variola, generally in the most modified form. Some of these had no cicatrix of their Vaccination, and in others the cicatrices were indifferent, or were only one or two; but of 57 who had three or more good cicatrices, only 2, or 3·5 per cent. had had small-pox. Of 82 medical men who had had small-pox, most of them by inoculation, in infancy—3 or 3·6 per cent. had contracted variola in later life.[2] These observations are strongly confirmatory of Jenner's anticipations as to the relative protective value of Vaccinia and small-pox.

2. The facts showing the power of Vaccinia in modifying small-pox, if it should happen to be subsequently contracted, and of disarming it of its terrors, are so ample that it is difficult to know whence to select examples. No epidemic of small-pox has occurred in any climate since the introduction of Vaccination without affording the most abundant evidence of it. While the mortality of natural small-pox is seldom below 20 per cent. and often amounts to 30 and 40 per cent. of the attacks, the death-rate among the vaccinated (taken indiscriminately and without regard to the quality of their vaccina-

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It will be seen that the *recent ratio of cases in epidemic years* has not exceeded *the average of ten years*, 1837–1846. This improvement is no doubt mainly due to the closer scrutiny now given on the admission of recruits to the evidences of their protection against small-pox, and to the performance of revaccination. As regards the relative protective value of variola and Vaccinia the improvement is significant, for the relative number of soldiers depending for their protection on Vaccinia is much greater now than before.

[1] *Lancet*, vol. xxxvi.

[2] Seaton, on the Protective and Modifying Powers of Vaccination.

tion) is rarely known to exceed 7 per cent. and is more frequently 3, 4, and 5 per cent. It will have been noticed, in the table above given of the cases of small-pox among the soldiers in the United Kingdom in the four years 1859-62, that of 430 cases of the disease only 24 were fatal, or 5½ per cent. In observations which, on account of the large scale on which they were made, are of great value, viz. those made for twenty-one years in Bohemia on four millions of people, it was found that the death-rate among vaccinated persons who happened to contract small-pox was $5\frac{1}{16}$ per cent. while the death-rate among non-vaccinated persons when they contracted small-pox was 29⅘ per cent.[1]

But the observations which outweigh all others in value, on account of the extreme accuracy and precision with which they have been made, are those which Mr. Marson has collected by thirty years' labour at the Small-pox Hospital. In this hospital above 15,000 cases of small-pox have during that time been under his personal care, and all particulars respecting them have been carefully recorded: and it has been found that while the unvaccinated have died at the rate of above 35 per cent., the vaccinated have died at the rate of only 6½ per cent.

But Mr. Marson's observations do far more than establish in a general way, in concurrence with others, the modifying power of Vaccination. They have a merit peculiarly their own. They show conclusively that *the degree of modifying power is in the exact ratio of the excellence and completeness of the Vaccination as shown by the cicatrices;* in other words, that it is directly as the amount of vaccine-marking and as the character of the marks. The subjoined table will show this at a glance, better than any detailed statement:[2]—

Classification of Patients affected with Small-pox.	Number of Deaths per cent. in each class respectively.
1. Unvaccinated	35
2. Stated to have been vaccinated, but having no cicatrix	23·57
3. Vaccinated—	
a. Having one vaccine cicatrix[3]	7·73
b. Having two vaccine cicatrices[3]	4·70
c. Having three vaccine cicatrices	1·95
d. Having four or more vaccine cicatrices	0·55
α. Having well-marked cicatrices	2·52
β. Having badly-marked cicatrices	8·82
4. Having previously had small-pox	19

So that while the average of vaccinated persons, if they should ever contract small-pox, have about one-sixth of the chance of having it fatality which is run by those who have not been vaccinated at all; some of them, from bad Vaccination, incur in fact one-third of that risk, while on the other hand others, thoroughly well vaccinated, incur less than one-seventieth part of it. In regard therefore to the expectation of any case of small-pox turning out badly, the question is not merely whether the patient has been vaccinated or not, but also *how* he has been vaccinated.

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[1] Simon, op. cit. p. xxvii.

[2] See also the article on Small-pox. The deaths from superadded disease have been deducted both from the unvaccinated and the vaccinated.

[3] In these classes the influence of quality of cicatrix was remarkably seen. In class *a* (with one vaccine cicatrix), among cases in which this was well marked, the death-rate per cent. was 3·83; among cases in which it was badly marked the death-rate was 11·91. In class *b* (two vaccine cicatrices), among cases in which these were well marked, the death-rate was 2·32; among cases in which they were badly marked it was 8·34.

These invaluable observations form the basis on which Vaccination should always be conducted. Nor, relying as we must do upon them, is it too much to affirm that no practitioner will have done his duty in any case in which he is called upon to vaccinate, unless, besides all requisite precautions with regard to the genuineness of the lymph employed and the means of insuring success, he has also taken care to vaccinate sufficiently, *i.e.* to produce, so far as in him lies, four or five genuine good-sized vesicles, such as result from separate punctures, or if vaccinating otherwise than by separate puncture, to produce equivalent local results.

Observations made by Dr. Buchanan and myself, during the epidemic of small-pox in London in 1863, on upwards of 50,000 children in various national and parochial schools, workhouses, &c., showed from another point of view the necessity of having regard to the quality and amount of Vaccination in estimating its protective value against small-pox. Some of the children examined had never been vaccinated: the large majority had been vaccinated in various manners and degrees. Of every 1,000 children without any mark of Vaccination we found that no fewer than 360 had scars of small-pox; while, of every 1,000 children who had evidence of Vaccination, only 1·78 had any such traces. And, on further classification with reference to the *kind* of Vaccination, we obtained the following results:—

Classification of Children examined.	Proportion marked with Small-pox per 1,000 Children in each class respectively.
1. Having no vaccine marks	360
2. Vaccinated—	
a. Having one vaccine cicatrix	6·80
b. Having two vaccine cicatrices	2·49
c. Having three vaccine cicatrices	1·42
d. Having four or more vaccine cicatrices	0·67
α. Having cicatrix or cicatrices of bad quality	7·60
β. Having cicatrix or cicatrices of tolerable quality	2·35
γ. Having cicatrix or cicatrices of excellent quality	1·22

On taking the extremes, it appeared that of children having four or more perfect vaccine marks, only 0·62 per thousand had any trace of small-pox, while of those who had a single bad mark of Vaccination, 19 per thousand were scarred by small-pox. As against small-pox therefore of such extent as to leave any traces, the best Vaccination had been upwards of thirty times as protective as the worst.[1] But this numerical statement is far from expressing the whole difference;

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[1] Sixth Report of Medical Officer of the Privy Council, pp. 91–2.

for the marks small-pox had left on the vaccinated, and particularly on the well-vaccinated, were for the most part very slight—the cases being quite exceptional in which there was anything approaching to disfigurement; while of the unvaccinated a very large proportion were seriously marked and disfigured. Many of them were really hideous to look at, and in several the small-pox had left permanent blindness or deafness.[1]

The protective power of Vaccination against small-pox extends to every race of mankind, and is seen in every climate and in every part of the habitable globe. Wherever small-pox has been known to occur, exemption from attack has been the rule among the vaccinated, the exception among the unvaccinated. Abundant illustrations of this protective value of Vaccination in hot climates will be found in Dr. Kinnis's Report on Small-pox in Ceylon in 1833–34,[2] in information collected by myself from the East and West Indies,[3] in the Annual Reports on Vaccination in the Bombay Presidency, &c. &c. And whenever in these climates vaccinated persons have contracted small-pox, it has usually been in the mild and modified form observed in this country. Thus, in an epidemic in the Mauritius, in which the mortality among unvaccinated patients was 42·7 per cent., it was found that of such vaccinated persons as contracted the disease, only 7 per cent. died, the greater part of these having unsatisfactory marks: and in the Ceylon epidemics reported by Dr. Kinnis, who, in his observations, noted in all instances the quality of the Vaccination, it was found that, while those who were known for certain never to have been vaccinated died at the rate of 41·5 per cent., and those who could give no account of themselves, or professing to have been vaccinated had no marks or bad marks, died at the rate of 26·1 per cent., persons having satisfactory marks of Vaccination died at the rate of only 1·6 per cent. Of 203 fatal cases, in fact, which occurred during these epidemics, only three were in persons having satisfactory marks of Vaccination, and two were in persons who had before had small-pox. In like manner in an epidemic in Jamaica in 1851, Drs. Bowerbank and Turner lost of 477 unprotected cases 75, but of 120 vaccinated patients only 4. Both the protective and modifying power was observed in the negroes and half-castes, as completely as in the white population.[4]

In consequence of the remarkable power of Vaccination in protecting against small-pox, and the adoption of the practice universally by educated people and in annually increasing proportion by the population at large, the present average death-rate from small-pox is scarcely in any European country one-tenth part, and in those countries in which Vaccination has been most carefully carried out it is much less than one-tenth part, of what it was at the end of the last century. Thus in Sweden, where before Vaccination was discovered the average annual death-rate from small-pox was 2,050, out of every million of population, during the forty years 1810-50, it was but 158; in Westphalia, where the small-pox death-rate used to be 2,643, it was from 1816-50 only 114; in Bohemia, Moravia, and Austrian Silesia, it has been reduced in like manner from 4,000 to 200; in Copenhagen, from 3,128 to 286; and in Berlin from 3,422 to 176.[1] And although our own country has been, at all events until lately, behind most others in Europe, as to the extent to which Vaccination has been adopted,[2] and behind many as to the completeness with which it has been performed,[3] yet the small-pox death-rate of England and Wales, which at the close of last century was estimated by Dr. Lettsom and Sir Gilbert Blane at not less than 3,000 per million of population, was for the average of the years 1841-53 only 304, and on the average of the succeeding twelve years 1854-65 has, notwithstanding three very severe epidemics, fallen as low as 202. The following table, exhibiting the small-pox mortality in England at various periods, not only shows the influence of Vaccination in the prevention of small-pox, but is of interest also as illustrating the value of legislative and administrative action in diffusing the blessings of that practice:—

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[1] One of the most remarkable illustrations of the protective power of Vaccination I ever met with was in a ragged industrial school at Hull—a really ragged school in which the children were of the very lowest class. Of 170 children in the school 33 had no mark of Vaccination, and 30 of these had marks of small-pox, most of them being greatly disfigured by it. Of the 137 having marks of Vaccination, only one had marks of small-pox, and these were very slight. Before detecting them I had already called the master's attention to the unsatisfactory character of the vaccine marks on the boy's arm.

[2] Report on Small-pox as it appeared in Ceylon in 1833-34, by J. Kinnis, M.D. 8vo. Colombo, 1835.

[3] Board of Health, Papers relating to the History and Practice of Vaccination, 1857, p. 139.

[4] Ibid.

Periods compared.	Annual deaths by Small-pox in England and Wales.	Annual rate per million of the Population.
1. Average of thirty years previous to introduction of Vaccination, estimated by Dr. Lettsom and Sir Gilbert Blane . .	—	3,000
2. Average of three years (1838–40[4]) when Vaccination had become to a great extent diffused, but before any public provision was made for its gratuitous performance.	11,944	770
3. Average of nine[5] of the years (1841–53) when public Vaccination was gratuitously provided, but Vaccination was not obligatory	5,221	304
4. Average of the twelve years (1854–65) during which Vaccination has been to a certain extent obligatory	3,967	202

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[1] Simon, op. cit. p. xxiii.

[2] Report of Small-pox and Vaccination Committee of Epidemiological Society, 1853.

[3] Marson, Medico-Chir. Trans. vol. xxxvi.

[4] The present system of registering deaths commenced only in 1837.

[5] During the years 1843-46 causes of death were not distinguished in the Reports of the Registrar-General.

With such proof of the protective value of Vaccination, it might well indeed be a matter of astonishment that small-pox should still annually make such ravages amongst us, if we had not ample evidence of the extent to which, through ignorance and apathy, and to a certain degree through prejudice not yet eradicated, the practice of Vaccination has been neglected, as well as of the imperfect and insufficient way in which the operation has not unfrequently been performed. The inquiries made, under the direction of the Government, from 1860 to 1864, by Drs. Stevens, Buchanan, Sanderson, and myself, into the state of Vaccination throughout England, showed that the universal performance of Vaccination in early infancy which is indispensable for the effectual protection of the community from small-pox was so far from being attained, that the proportion unvaccinated, even among children old enough to be in attendance at public infant schools, exceeded 13 per cent. Now, it is in the young unvaccinated portion of the population that the small-pox mortality chiefly occurs. Of the nearly 4,000 deaths from this disease which are on an average still recorded every year in England, 56 per cent. are in children under five years of age, and as much as 70 per cent. in children under ten years of age.[1] We can have no hesitation in saying that in all the fatal cases at this early age there must, with very rare exceptions, have been neglect of Vaccination; for when that operation has been performed, even with the effect of raising a single vesicle only, subsequent death from small-pox in *childhood* very seldom indeed occurs.[2] We know further that of the mortality above ten years of age a very large proportion takes place in persons in whom Vaccination had never been performed. So that an estimate which should ascribe four-fifths of the present mortality from small-pox to the omission of Vaccination would most certainly be very much below the mark. Many, however, of the deaths from small-pox which take place after puberty—and there are on an average above 1,000 deaths annually from this cause in England in persons over fifteen years of age—are (especially at epidemic periods) in individuals who had been vaccinated, and who believed themselves protected against small-pox. But as regards these, there arises the further question, *how* had they been vaccinated? Now, though we cannot, of course, answer this question as regards the individuals, we may fairly apply to the group the observations made in the Small-pox Hospital on fatal cases of small-pox in persons believed to have been successfully vaccinated. Of 402 such fatal cases, occurring in 20 years, 101 exhibited on their arms no evidence whatever of

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[1] Deaths in England from Small-pox, at different ages, for the nine years 1855–63:—

All ages.	Under 1 Year.	1—2 Years.	2—3 Years.	3—4 Years.	4—5 Years.	Under 5 Years.	5—10 Years.	10—15 Years.	15—25 Years.	25—35 Years.	35 Years & upwards.
30,707	7,334	3,370	2,666	2,152	1,732	17,254	4,078	1,169	3,552	2,422	2,232

[2] Gregory, Marson, &c. as quoted in "Handbook of Vaccination," pp. 224–5.

having ever had effective Vaccination; 277 had but one or two vaccine marks, and these, in 191 of them, were of imperfect character; 16 were in persons who had three cicatrices; and only 5 in persons who had been vaccinated in the way which has been shown to be the most protective—of which 5, two did not die of the small-pox, but of concurrent or superadded disease.[1] We may be sure then that in only a few of those who die from small-pox after Vaccination in England, the Vaccination had been done in the best way.

In the official inquiries above referred to, in the course of which the arms of nearly half a million vaccinated children were examined, evidence was obtained of the great extent to which imperfect or insufficient Vaccination had heretofore prevailed in England; taking the country throughout, not more than one child in eight was found to be so vaccinated as to have the highest degree of protection that Vaccination is capable of affording; not more than one in three could, on the most indulgent estimate, be considered as well protected; while in more than one in four the Vaccination had been of a very inferior kind indeed, resulting in marks of imperfect character, or in only one or two marks of merely passable character.[2] These imperfections were mainly traceable to the following causes:—(1) the frequency with which practitioners, instead of attempting fully to infect the system, had been satisfied with insertions of lymph, sufficient to produce only one, two, or three ordinary vesicles; (2) the want of due attention to the *selection* of the lymph used in vaccinating; (3) carelessness and clumsiness in the performance of the Vaccination, so that, if the operation did not wholly fail, it very frequently resulted in a less degree of effect than it had been the aim of the operator to produce; and (4) the great and unnecessary extent to which the use of preserved and conveyed lymph was substituted for the Vaccination direct from the arm, which should be the rule of all vaccinators.[3]

It is satisfactory that these, the chief causes of imperfection, are of a kind for which we have in future an obvious remedy (and which, it may be added, are now rapidly being remedied), in the proper practical instruction of vaccinators, in the employment of more care and attention in vaccinating, and in better arrangements for transferring lymph. Other causes of imperfect Vaccination which are quite independent of the vaccinator, though far less widely operative than those just enumerated, must not, however, be overlooked: as, something particular in the child's constitution or condition at the time of Vaccination, which even the most experienced vaccinator may have been unable to detect; aberrations of lymph—rarely indeed, but still occasionally, met with—occurring under undefinable conditions, of which the first manifestation to the vaccinator has been the unsatisfactory result; the carelessness of parents in allowing the vaccine vesicles to be disturbed in their course and the crusts to be prematurely removed.

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[1] See also the article on Small-pox.

[2] These observations were made on children most of whom had been vaccinated by public vaccinators, but a large number of whom had been operated on by private practitioners, and, without affording statistical evidence of the fact, they left a strong impression that, as a rule, the latter were less well vaccinated than the former. My own experience has satisfied me in other ways that many in the upper and middle classes in England have been very imperfectly vaccinated—the chief reason why small-pox is so much less met with among them than it is among the lower classes being that they are so very much less exposed to it.

[3] For evidence in detail see Reports of Medical Officer of Privy Council, iii.—vii. It is beyond the limits permissible for the present article to enter into an inquiry into the subject of the best arrangements for the performance of Vaccination and for maintaining stocks of active lymph: but it is a subject of the utmost importance, and one which I have endeavoured to treat fully in my "Handbook of Vaccination," chap. vii.

It has been contended that, apart from imperfections in the mode of conducting Vaccination, the vaccine lymph itself necessarily degenerates by repeated transmissions through the human body, and loses something of its infective and protective power.

The hypothesis that lymph would deteriorate by mere transmission through human bodies was started in the very earliest days of Vaccination. Jenner thought the notion a very improbable one, but could only then say of it that time was necessary to determine the question. But after many years' experience he felt himself quite justified in pronouncing the hypothesis as groundless. Drawing, as he never failed to draw, the broad and most important distinction between such deterioration of lymph as may result if in continuous vaccinations due care be not taken in selection of the lymph employed, and the doctrine of inevitable deterioration by mere transmission: he remarked, writing in 1816, that lymph in passing even from one individual to another might undergo a change which rendered it unfit for further use, but that the notion of necessary degeneration was a conjecture "he could destroy by facts:" and he referred, in proof, to the vesicles he was then producing, which, he says, "are in every respect as perfect and correct in size, shape, colour, state of the lymph, the period of the appearance and disappearance of the areola, its tint, and finally the compact texture of the scab, as they were in the first year of Vaccination; and to the best of my knowledge the matter from which they are derived was that taken from a cow about sixteen years ago."[1] Numerous trustworthy observers, who had watched the vaccine disease at the introduction of Vaccination, on comparing what they had then seen with the effects produced by lymph of the earliest stocks, after a lapse of thirty or forty years, were unable to detect the slightest difference either in the character or course of the vesicles. And the same appears certainly to be the case at the present day. Having for the last thirty years been a close observer of the vaccine vesicle, and having during the last nine years enjoyed such opportunities of witnessing the practice of Vaccination in the hands of various vaccinators, and of noticing the results of Vaccination in the cicatrices left on the arms of individuals vaccinated, as have scarcely perhaps fallen to the lot of any other person, I can confidently affirm that the vaccinators of the present day who are masters of their craft do their work as surely with ordinary long-humanized lymph and infect their patients as completely as the earlier vaccinators did, producing vesicles which in character and course differ in nothing from the description that Jenner has given us, and which leave cicatrices as perfect as those which I have seen on the arms of persons who had been vaccinated by Jenner himself or by his well-known contemporary Dr. Walker.[1] Whatever has been found unsatisfactory in the hands of other vaccinators, whether as regards the course of the vesicles or the character of the cicatrix, was fairly traceable to the causes which have been already stated, and especially to looseness and carelessness in the selection of lymph: and though I have sometimes found it desirable to recommend a vaccinator to change the stock of lymph he was employing, I have always felt it enough to take care that the stock substituted was good active lymph without troubling myself as to the time that had elapsed since it had come from the cow. It is quite true that the earliest transmissions of lymph from the cow to the human subject have usually a peculiar intensity of local irritative effect. But this result (which is so far from being an advantage that it is often found to need controlling)[2] is generally lost in a few transmissions, often indeed in one or two transmissions; besides that different primary stocks are found to differ much as to the local effect they thus produce.[3] Ceely, whose testimony on this subject is of the utmost importance, because he, more than any other living inquirer, has studied the natural disease in the cow, and has experimented on its transference to the human subject, entirely disclaims belief in the superior protective efficacy of lymph thus recently transferred over active humanized lymph.[4] Several practitioners residing near the Bridgewater Level, and in the Vale of Gloucester (in which districts the natural cow-pox is still not unfrequently seen in the dairy farms), stated a few years ago to Dr. Sanderson that they had inoculated lymph direct from the cow with success; but all agreed that, after the first or second transmission, the results did not differ from those of ordinary Vaccination, either in the character or progress of the vesicles. It is in truth not to the cow, but to adequate care and skill on the part of vaccinators in the selection of the children and vesicles from which lymph is taken, that we must look for maintaining stocks of active lymph.

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[1] Letter in Baron's Life, vol. ii. p. 398. M. Bousquet appears to have overlooked this passage. See his "Nouveau Traité de la Vaccine," p. 399.

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[1] See Fourth Report of Medical Officer of Privy Council, p. 64.

[2] To one unpleasant result frequently met with in early vaccinations from the cow, Mr. Ceely has especially called attention in a recent communication; it is the occurrence of a special vesicular vaccine eruption called by the Germans "Nachpocken," which causes a good deal of temporary disfigurement and annoyance, and sometimes, when copious, severe and even dangerous symptoms. (See Brit. Med. Journ. Jan. 7, 1865.)

[3] Handbook of Vaccination, chap. x.

[4] Observations on Var. Vacc. in Trans. of Prov. Med. Assoc. vol. viii.

[5] The important point, it seems to me, is that vaccinators should not be induced to look to anything extrinsic and inevitable for an explanation of deteriorations which, if they occur at all, may be more properly ascribed to want of skill and care somewhere. So long as this is kept in mind, there can of course be no objection to the introduction at any time, when opportunity offers, of fresh stocks; but those who are about to under-

Facts are equally at variance with the supposition that transmission through human bodies causes vaccine lymph to lose anything of its *protective* power. Persons vaccinated with lymph direct, or only a few removes from the cow, and others vaccinated with long-humanized lymph, have been submitted to the test of variolous inoculation; but the result in each class of cases has been the same. This experiment in fact is made to hand in a large scale in every epidemic of small-pox. In each such epidemic—no matter at what period since the introduction of Vaccination—it has not been the persons vaccinated with the then current lymph, the lymph furthest from the cow, but those vaccinated some sixteen, twenty, or twenty-five years before, with lymph so much the nearer to the parent source, who have been found to be the chief sufferers from post-vaccinal small-pox.

Another subject for consideration in reference to the protective power of Vaccination is, its permanency in the individual. It has been often said that it is undoubtedly a protection, but for a limited time only; that it wears out by age, and requires successive renewals. Although this theory is sometimes thus broadly advanced, the permanency of the protection which a single efficient Vaccination gives against small-pox is so completely established as the law of the human economy, that we need only deal with it as offering an explanation of *that proportion of cases* in which small-pox is met with in those who have been vaccinated. As regards these, we may observe in the first place that at times when epidemic small-pox prevails, and especially under circumstances of great exposure, as where children are living and sleeping in the same room, perhaps sleeping in the same bed, with a case of small-pox, the disease is sometimes met with in very young children, and occasionally very shortly after Vaccination. Even so early as the year 1806, Willan was able to record numerous cases in which a very trivial eruption, but of true variolous character, appeared in children at intervals which varied from five months to seven years after Vaccination had been performed. Similar examples have been met with in every epidemic down to the present time. But this eruption is generally so slight (except where the Vaccination has been spurious, or ineffective) as to attract little attention, or only to attract observation on account of its amazing contrast with the variola of unprotected children. After puberty small-pox is met with in vaccinated persons more frequently, and though no doubt generally modified, is, if there have been any imperfection in the original Vaccination, in not a few cases severe and even fatal; even after the most complete Vaccination, cases are met with, some of which are severe, and a very few—not much more than the half of 1 per cent.—fatal. It has been found, however, in the experience of the army, in that of the Small-pox Hospital, and in practice generally, that cases of small-pox after Vaccination occur chiefly between the ages of fifteen

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take the transplantation of lymph may meet with some difficulties, and would do well to study Mr. Ceely's admirable "Observations on the Variolæ Vaccinæ," in Trans. of the Prov. Med. Assoc. vols. viii. and x.

and twenty-five, and that after the age of twenty-five they sensibly diminish. Thus, while in the army the deaths from small-pox among soldiers under twenty years of age were 3·4 per 10,000 of aggregate strength, and those among soldiers from twenty to twenty-five years old 3·1 per 10,000; the deaths from this cause among the soldiers above twenty-five years old have been but 1 per 10,000. And from the observations of Professor Heim, on 1,055 cases of small-pox in vaccinated persons, examined not in relation to the age but specially as to the number of years that had elapsed since Vaccination, we find that while the average annual number of cases that occurred in the first twelve years after Vaccination was twelve, and in the next thirteen years was over fifty-one; the average for the following ten years was under twenty-five. The real explanation appears to lie not in the weakening influence of time, which would be a progressively increasing influence, but rather in certain disturbing influences of which, no doubt, puberty is the chief.

The liability of persons to renew their susceptibility to small-pox is, unquestionably, very much a matter of diathesis. It is frequently met with in various members of the same family; and I know of many instances in which, under the same circumstances, other members of these families have suffered from second attacks of small-pox.

Revaccination.—The numerous instances in which, from whatever cause, the protection of Vaccination has proved insufficient, have led to the very frequent adoption of late years of revaccination. By many this practice is looked upon as only called for or useful where there has been some defect in the primary Vaccination; but there is reason to believe that it has a use beyond this—that it extinguishes that renewed susceptibility to small-pox which, it has been already pointed out, occurs in an indeterminate proportion of persons after even the most perfect Vaccination.

When a child has unfortunately been imperfectly vaccinated, no doubt the best thing that can be done is to vaccinate it again. But the chance revaccination may afford of correcting original imperfection must never be held for a moment to absolve any practitioner from the pains he is bound to bestow to make his original Vaccination full and complete. For what daily happens is this:—A child is vaccinated and takes badly; either at once, or at some no distant period, it is vaccinated again, and perhaps a third time, and cannot be made to take; it grows up, gets small-pox, and very likely dies. Or this happens: The parents are directed to bring it again at some specified time, and fail to do so, and the same fate as in the other case befalls it. Take it at the best, an originally imperfect or incomplete Vaccination is a very great misfortune.

But supposing it to have occurred, how is the practitioner to act? Is he to revaccinate at once, or is he to wait till puberty when the chief danger of insufficient Vaccination manifests itself? He must be guided in determining partly by the degree of imperfection, and partly by the liability to exposure to small-pox. If the Vaccination has been spurious, irregular, or disturbed in such a way as to divest it of protective power, he would, unless there were something in the child's then state of health which might have been the cause of these imperfections or irregularities, at the earliest opportunity vaccinate it again; if anything were found amiss with the child, he would of course wait till that was corrected. Very likely the Vaccination would not take, for quite spurious Vaccination will often prevent real Vaccination from taking effect afterwards. But the chance must be given, and given with every care to produce effect. If such should result, even though it be only the ordinary spurious effect of a revaccination, he will have done all he can do, and need recommend no further proceedings till puberty—at all events unless there should arise some immediate danger of small-pox. But if there be no local evidence that the lymph applied on the revaccination had been absorbed, the operation should be repeated at intervals until he is satisfied that the child is, for the time at all events, insusceptible. Supposing, however, the result of the primary Vaccination have been one of insufficiency rather than of imperfection,—that is, if a single vesicle have risen instead of the four or five it was desired to produce, but that vesicle has run its course perfectly,—I do not usually recommend, except under circumstances of danger, any further Vaccination till the child grows up.

The same rule guides me in judging from the cicatrices left on the arms of young children whether revaccination is called for. If these be decidedly imperfect in character, I advise the parents to seek revaccination with as little delay as possible; but if the cicatrices be only deficient in number, or if the character, though less strikingly good than it might be, is yet genuine, I hold the child to be pretty safe up to the age of puberty, but strictly enjoin its revaccination then.

The occurrence of a case of small-pox in a house leads to a stricter rule as regards all the inmates. Those who are past or approaching puberty should, except such as have already since puberty been successfully revaccinated, be revaccinated at once; and those under puberty who have two or more thoroughly characteristic marks should not be meddled with; the children whose marks are not thoroughly good, or who have but a single good mark, should be revaccinated. In a crowded court this course should not be limited to the house in which the small-pox appeared, but extended to each house. If, on the outbreak of an epidemic of small-pox, this plan were uniformly adopted, together, of course, with the immediate Vaccination of all who in the house or court were found unvaccinated, there can be very little doubt the epidemic might be cut short, and very certainly indeed the occurrence of fatal or severe cases of small-pox would be all but entirely prevented.[1]

Revaccination about, or after, puberty is of extreme importance when the original Vaccination has been anything short of Mr. Marson's highest class, and is necessary in proportion as it falls short of it: but it seems also certain that those whose original Vaccination has been complete may derive additional security from a revaccination at, or after, this period of their lives. We have already seen that a certain proportion of the most thoroughly vaccinated contract small-pox, though no doubt with comparatively little danger, after growing up: as many as 367 such cases were admitted to the Small-pox Hospital in the great epidemic of 1863. But after effectual *revaccination*, small-pox, even in its most modified form, is found very rarely, or scarcely ever, to occur. Thus, Heim found that in five years there occurred among 14,384 revaccinated soldiers in Wirtemberg, only one instance of varioloid, and among 30,000 revaccinated persons in civil practice only two cases of varioloid, though during these years small-pox had prevailed in 344 localities, producing 1674 cases of modified or unmodified small-pox among the not revaccinated, and in part not vaccinated, population of 363,298 persons in those places in which it had prevailed. In the Prussian army, since the introduction of systematic revaccination of all, the annual deaths from small-pox (which at one time were 104) have not averaged more than 2; and on analysis of 40 fatal cases that occurred in twenty years, it appeared that only 4 were in persons who were said to have been successfully revaccinated.[1] Other national experience might be referred to, but it will be better to have recourse once more to Mr. Marson's very precise statements. In the thirty-two years and upwards that he has been connected with the Small-pox Hospital, no nurse or servant has taken small-pox, he having taken care always to revaccinate them on their coming to live in the hospital; and at a time when a large number of workpeople were employed for several months about the hospital, most of whom consented to be revaccinated, two only were attacked by small-pox, and these two were amongst the few who were not revaccinated.

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[1] See a remarkable illustration of this in Third Report of Medical Officer of Privy Council, p. 50; and see also illustrations in Report of Small-pox and Vaccination Committee of Epidemiological Society, 1853.

These broad facts, while they show the great importance of the practice of revaccination, attest at the same time the utter uselessness and folly of repeating this operation again and again in the same individual, as seems to have become a recent fashion, whenever epidemics of small-pox arise. One thoroughly good primary Vaccination to start with, and one careful revaccination after puberty, so conducted as to give evidence that the lymph was absorbed, are all that is necessary for the complete protection of the population against small-pox. No doubt by the first Vaccination, nine out of ten are perfectly well and permanently protected; but who can predicate of any individual whether he is one of the nine, or the exceptional one?

The revaccination, however, must be done with all the care that should be employed to secure the success of a primary Vaccination; and the practitioner should always endeavour to get some evidence that the lymph is absorbed. In many cases, however, even after repetition of the Vaccination, this will not happen; and in these cases a further attempt may very properly be made at some future time. Revaccination should always be done by preference when it can be done leisurely, and as part of the ordinary work of Vaccination, and not under the alarm and influence of panic.[1] The wholesale and sham revaccinations, which would under such circumstances appear to have been practised of late, will tend much, it is to be feared, to bring the practice into disrepute and contempt.[2]

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[1] Simon, op. cit. pp. xxxv. xxxvi.

The following table, showing the results of revaccination in each 1,000 individuals revaccinated in the Wirtemberg army in 1831-35, and in our own army in 1861, will give some indication of the local results that may be expected from the performance of revaccination in adults:—

Persons in whom the Revaccinations were performed.	Degree of success of Revaccination.	In those who bore marks of previous Small-pox.	In those who bore good marks of previous Vaccination.	In those who bore doubtful or imperfect marks of previous Vaccination.	In those who bore no marks of previous Vaccination or Small-pox.
Wirtemberg Army, 1831–5 (13,861 cases)	Perfect	319·5	310·4	280·7	337·3
	Modified	248·1	280·5	259·	191·1
	None	432·3	409·2	460·4	471·6
		1000	1000	1000	1000
Soldiers in Brit. Army, not recruits, in 1861 (2,053 cases)	Perfect	451·4	484·6	236·8	326·
	Modified	159·6	157·4	505·3	277·5
	None	389·0	358·0	257·9	396·5
		1000	1000	1000	1000
Recruits in Brit. Army in 1861 (4,395 cases)	Perfect	345·5	407·3	461·3	527·3
	Modified	266·8	240·8	301·3	202·6
	None	387·7	351·9	237·4	270·1
		1000	1000	1000	1000

A perfect local result following a revaccination is constantly appealed to by practitioners as evidence that the person in whom it was developed was liable to take small-pox, or, at all events, more liable than those in whom imperfect or no results followed. But this conclusion appears to me by no means warranted. If it were, these curious results would follow, that (taking as our guide the observations in the Wirtemberg army) 319 out of 1000 persons having had small-pox, 310 out of 1000 who had been well-vaccinated, and only 281 out of 1000 who had been ill-vaccinated, were in present danger of taking small-pox; and of the soldiers (not recruits) in our own army, 451, 485, and 237 would represent the ratio in the three classes respectively, which is clearly a *reductio ad absurdum.* We cannot indeed, that I can see, draw from the local phenomena of revaccination any inferences whatever as to the state in which the revaccinated person was as to liability to small-pox. Jenner himself, indeed, pointed this out in his first treatise, and showed that the natural cow-pox might be induced again and again in persons who, being protected against variola by their first attack of Cow-pox, could not be variolated either by inoculation or by exposure, and also that Cow-pox might be made to take on those who had had small-pox.[1] The utility and necessity of revaccination stand not upon any speculative reasoning from the local effects it produces, but upon broad grounds of observation and experience.

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[1] Under these circumstances the demand for revaccination often becomes so great that it is difficult to find lymph to meet it. I have known revaccination-lymph used under these circumstances for performing other revaccinations, and very bad arms resulting.

[2] See Sixth Report, Medical Officer of Privy Council, p. 113.

IV. Relations of Variola and Vaccinia.—Jenner believed the cow-pox of the cow and the small-pox of the human subject to be essentially the same disease, as he implied when he denominated the former Variolæ Vaccinæ: he further believed they had a common origin—the grease of the horse. He did not himself perform any inoculation of cattle with the lymph of human variola. But, as early as 1801, Gassner, of Günsburg, by inoculating eleven cows with small-pox matter, produced on one of them vesicles having all the character of vaccine vesicles, and from which a stock of genuine vaccine lymph was obtained. Another successful variolous inoculation of cows, at the Veterinary College at Berlin, is referred to by M. Viborg of Copenhagen so early as 1802, but no details are given respecting it. In 1830, Dr. Sonderland, of Barmen, stated that he had infected cows with the variolous contagion by enveloping them in blankets taken from the bed of a patient who had died of small-pox, and also hanging the blankets up round the head of the animal that it might breathe the effluvia arising from them. The cows, he says, in a few days manifested the symptoms of Cow-pox, and lymph taken from them produced genuine vaccine vesicles in the human subject. Dr. Sonderland's experiments were repeated in India by Mr. Macpherson, in this country by Mr. Ceely of Aylesbury, and in various places abroad, without success. But Mr. Ceely was able, by the much more satisfactory process of direct inoculation with small-pox virus (in February 1839), to induce vesicles in two out of three sturks operated on, and with lymph taken from these to vaccinate many children, from whom a regular lymph-stock was continued.[2] In 1840, Mr. Badcock of Brighton, without previous knowledge of Mr. Ceely's experiments, succeeded also in variolating the cow, and deriving thence a stock of genuine vaccine lymph: and since then he has, by inoculating cows with the lymph of human variola, raised stocks of vaccine lymph for use on no fewer than thirty-seven separate occasions. The common origin, then, of small-pox and cow-pox may thus be considered as established.

The case, as regards the grease, appears to stand thus: the disease really known as grease has nothing to do with cow-pox or small-pox; but the horse is subject at times to a true equine pox, which is precisely of the same kind as the small-pox in man and the cow-pox in the cow. This disease is met with as an epizootic, particularly when cow-pox is epizootic among cows and small-pox is epidemic. The equine matter used by Jenner himself,[1] and that used by Sacco and others, for the purposes of Vaccination, were derived from this equine pox; and on various other occasions equine lymph has been employed.

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[1] Obs. on the Variolæ Vaccinæ, pp. 21, 22, and p. 51; Continuation, &c. p. 25.

[2] Obs. on Var. Vacc. in Transactions of Prov. Medical Assoc. vol. viii. Three years before, Dr. Thiele, of Kasan, in Russia, had made similar successful experiments (Henke's Zeitschrift, t. xxxvii. h. 1), which were not known to Mr. Ceely, and, in fact, were not published till 1839.

V. Alleged Dangers of Vaccination.—Space renders it quite impossible for me to enter into the consideration of certain objections that have been urged from time to time to the general utility of Vaccination, or have been thought to prove that it was injurious: such as the displacement-of-mortality theory of M. Carnot, the allegations that scrofula and typhoid fever have become more frequent in consequence of the introduction of the practice (being in fact, it has been said, "vaccinational varieties or introversions of small-pox"), and other like absurdities. The practitioner who desires to acquaint himself with all that has been brought forward on these subjects, will find the fallacies of the various statements thoroughly exposed, and the questions themselves finally settled, in the admirable memoir which Mr. Simon has prefixed to the Papers relating to the History and Practice of Vaccination, to which I have already so often referred. What is before us now to consider is of more limited scope, but of much greater real importance, and relates not to whether Vaccination is a proceeding we ought still all to adopt, but to the special care and precaution which should be brought to its practice. Is it possible in vaccinating to communicate accidentally other diseases—as cutaneous diseases, scrofula, or syphilis?

Reserving for separate consideration what has to be said regarding syphilis, I may state that the invaccination of cutaneous and scrofulous diseases, though a popular, has never been a professional belief. These diseases are met with constantly in infancy and childhood, as well in the unvaccinated as in the vaccinated, from the influence of various exciting causes acting on constitutional predisposition. I am not aware of any facts which prove, or even render probable, their greater frequency among vaccinated than among unvaccinated children of the same ages respectively. When eczema and other eruptions manifest themselves, as they may do, shortly after Vaccination has been performed,[3] this is held by some to be—and no doubt very generally is—a mere coincidence, and due in reality to one of the various eruption-producing influences to which children at the usual age for Vaccination are subject, such as teething, &c. But there are many medical men who hold, and with great probability, that in a part at least of these cases the Vaccination may itself have been, by the febrile action it set up, the *exciting* cause. No medical authorities believe in the transference of scrofulous and cutaneous diseases from one child to another by Vaccination. Parents, however, as Mr. Marson observes, "are unwilling to believe that there is anything constitutionally wrong in their offspring; and, when other diseases follow, Vaccination gets blamed for what is really and truly due to other causes." Hence, parental complaints that disease has been set up in this way are not unfrequent: but, as showing the prejudice under which such complaints are preferred, it may be worth while to state that though I have carefully investigated a great number of them, I have never yet in a single instance found that the child from whom the lymph was taken was suffering from the disease it was said to have imparted.

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[1] Baron's Life, vol. i. p. 254.

[2] The limits of this article do not allow me to enter further into this interesting subject, which is fully discussed in chaps. ii. and iv. of my "Handbook of Vaccination."

[3] Such eruptions as are part and parcel of the constitutional symptoms of Vaccination have been already treated of in Sect. i.

Those who have had most to do with the performance of Vaccination, on the one hand, and those who have been most concerned in the treatment of infantile disease, on the other, concur in the belief of the non-communicability of disease by Vaccination. Mr. Marson, in the performance of 50,000 vaccinations and more, "has never seen other diseases communicated with the vaccine disease, nor does he believe in the popular reports that they are so communicated."[1] Such also was the experience of the late Mr. Leese, whose opportunities of observation were scarcely, if any, less.[2] Sir W. Jenner stated some years ago that at University College Hospital and at the Hospital for Sick Children he had had, in six years, more than 13,000 sick adults and children under observation, and that in no case had he reason to believe, or even to suspect, that any constitutional taint had been conveyed from one person to another by Vaccination.[3] Dr. West's experience on 26,000 infants and children under his care in seventeen years is to the like effect; and in stating that he had seen nothing in that time to make him believe that Vaccination excites cutaneous eruptions in any but very exceptional cases, he referred such exceptional cases to a disposition in the children themselves, brought out by the Vaccination as it might have been by teething.[4] And Professor Paget, speaking from his large experience among children in the out-patients' room at St. Bartholomew's, and enumerating some of the causes which develop cutaneous diseases in young children, says, "Now, Vaccination may do, though I believe it very rarely does, what these several accidents may do; namely, by disturbing for a time the general health, it may give opportunity for the external manifestation and complete evolution of some constitutional affection, which, but for it, might have remained rather longer latent." "This is," he adds, "the worst thing that can with any show of reason be charged against Vaccination; even this can very seldom be charged with truth."[5]

Although the direct inoculability of the syphilitic poison from one human being to another distinguishes it remarkably from cutaneous diseases generally and from scrofula, I should still, but for certain recent occurrences which have excited much attention, and to which I shall immediately advert, not have thought it necessary to speak separately of the communication of that disease by Vaccination. It was indeed not only included with other diseases, but was specially so included, in the opinions of the distinguished practitioners whom I have just cited; Dr. West informing us that there had never come under his notice "any instance in which there seemed the slightest pretext for supposing that syphilis had been communicated to infants through the medium of the vaccine lymph," and Professor Paget that he does not remember "to have heard infantile syphilis ascribed to Vaccination, frequent as the instances of it (inf. syphilis) are among the out-patients." In the experience of Mr. Marson, Mr. Leese, or the National Vaccine Establishment of England, such a case has never been met with. And referring generally to the experience of practitioners at home and abroad, it may safely be said that there is scarcely a subject in medicine in which there has been, till within the last few years, a more general concurrence of opinion.[1] One broad general fact seemed to be, and still probably is, conclusive on the matter. In the seventy years that have passed since Vaccination was introduced, it seems certain that "if syphilis could be diffused by the vaccine lymph of children with an hereditary taint of that disease, this possibility must long ago have been made evident on a scale far too considerable for question."[2]

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[1] Papers relating to the History and Practice of Vaccination, p. 25.

[2] Seaton on Protective and Modifying Powers of Vaccination, p. 23.

[3] Papers relating to, &c. p. 75.

[4] Ibid. p. 146.

[5] Ibid.

Scientific authority unites with general medical experience to negative the possibility of the vaccinal communication of syphilis,—it being implied always of course that the vaccination is true vaccination, *i.e.* with vaccine lymph taken from a true Jennerian vesicle. Professor Paget states the pathological grounds for disbelieving the possibility of any such communication to be, (1) because *infantile syphilis* (which alone need be here considered), though conveyable in some instances by its own peculiar morbid products, does not render the blood of the patient capable of directly conveying the disease; and (2) because, if the blood of a syphilitic child could so modify the vaccine disease within it as that the vaccine lymph should be capable of conveying any other disease, there is every reason to believe that the vaccine vesicle formed in the diseased child would be modified in correspondence with the modified lymph. "All pathological researches," he observes, "accumulate the evidences of the constant correspondence between the material in the blood, on which each specific disease depends, and the morbid structure, by which each is manifested. Thus the transformations of the syphilitic poison are indicated in the successive external characters of the primary, secondary, and tertiary affections; the transformation of the scarlatina poison by its regular symptoms and its sequelæ. And so, if the vaccine virus were capable of any transformations besides those which mark its regular influence in each patient, such transformations, we may be sure, would be indicated by corresponding and evident changes in the vaccine vesicle. In other words, if the vaccine were changed into any other virus, there would be no vaccine vesicle."[1] The opinions of Hebra and other distinguished pathologists are to the like effect.

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[1] See replies of eminent members of the medical profession to the queries addressed to them (in 1856) by Mr. Simon.

[2] Simon, in Papers, &c. p. lxvi.

Direct experiments made on a large scale, at many times, and by many individuals, have led in every single instance to the same conclusion. M. Cullerier and other experimenters in France, especially M. Taupin, have taken lymph on purpose from syphilitic children, have vaccinated healthy children with it, and watched the result. In no instance has syphilis been communicated. Heim made similar experiments in Germany with the same result. It was found no more possible to produce syphilis by vaccine lymph taken from a syphilitic child, than it is to produce small-pox by lymph taken from vaccine vesicles on the arms of patients who are incubating, or suffering from that disease. This, it is well known, has been done hundreds of times, but never has small-pox been thus communicated or anything but a vaccine vesicle resulted.[2]

Cases had indeed been from time to time recorded in which it was believed or suspected that syphilis had been communicated from one person to another along with Vaccinia. But, in searching the literature of sixty years, notice can scarcely be found of a dozen occasions in which events of this kind were supposed to have happened, where the circumstances are stated with sufficient detail to enable us to form a judgment of the value of the evidence; and, in all of these, the details, when they were examined, were found so wanting in scientific precision, and so open to sources of fallacy, that the cases had been rejected as worthless for proof. So that about eight years ago the mind of the profession generally, never till then, so far as I am aware, very seriously disturbed, may be said to have been at rest on the subject. This quietude, however, was soon afterwards broken by a very circumstantial account of a singular outbreak of endemic syphilis at Rivalta, in 1861, traceable, it was said, to a vaccinal origin, and by one or two cases—especially by one which occurred in the wards of M. Trousseau, at the Hôtel Dieu in Paris, in 1861—which afforded, it was alleged, direct proof of vaccino-syphilitic inoculation. To the events then announced a new and adventitious interest was imparted by the bearing which proof of the occurrence of vaccino-syphilitic inoculation, if it could be afforded, would have on the doctrines then being sharply contended for by rival schools of syphilographers.[3] All the cases detailed from the earliest days of Vaccination were eagerly hunted up; the discussions which ensued caused a few fresh (alleged) cases to be recorded; and the evidence thus collected has been held by certain authorities in syphilis to establish that the inoculation of syphilis in vaccinating from a genuine vaccine vesicle, though of excessively rare occurrence, is yet possible, supposing that the child from whom the lymph was taken had, or was incubating, syphilis, and that some of the blood of the syphilitic child was inoculated along with the vaccine lymph. The allegation is not, it will be observed, of carelessly syphilizing instead of vaccinating, as by taking syphilitic matter instead of, or along with, vaccine lymph, in which cases we might expect, of course, syphilis only, or syphilis with Vaccinia, to result. It is that in the ordinary performance of Vaccination (the absence of syphilitic matter on the lancet being presupposed), syphilis may be accidentally implanted along with the vaccine. It is not contended, in face of the accumulated evidence to the contrary from pathological science, general experience, and direct experiment, that vaccine lymph would impart syphilis or any other than its own specific contagion. But it is said there may be twofold inoculation, and the communication may take place through the blood.[1] Lymph and syphilitic blood being inoculated together, each within its own period of incubation will produce its own specific results. The vaccine disease will first run its course, and this being over or approaching its end, the effects of the blood inoculation will manifest themselves. But even of the possibility of accidental communication in this way it appears to me that the very strictest proof, and a complete absence of every possible source of fallacy,[2] are demanded: (1) because we have hitherto been without any evidence whatever of the direct communication of *infantile* syphilis by inoculation of blood; (2) because inoculation of syphilis by blood of the adult is a matter of very great difficulty and very frequent failure, requiring always the exposure of a very large absorbing surface, while in the cases now brought forward the inoculation seems to be effected wholesale and by the minutest drop of blood; and (3) because in the numerous vaccinations which in the course of seventy years must have been done accidentally from syphilitic children, blood must often have been invaccinated, and in many of the experimental vaccinations from syphilitic children blood was purposely mixed, and yet no syphilis had resulted.

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[1] Papers relating to, &c. p. 139.

[2] Simon, op. cit. p. 43.

[3] A reader of the recent discussion in the Académie de Médecine will be surprised at the sort of jubilant tone with which the vaccinal inoculation of syphilis is hailed as giving the "dernier coup" to a certain "école syphilographique," &c. &c. as well as at the mere hearsay evidence on which facts are pressed into the controversy.

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[1] In experiments with the mixed viruses made by Sigmund, by Friedinger, and by Boeck, syphilis *only* was produced: there was no Vaccinia. In one of Friedinger's experiments, however, and in one out of many experiments by Baumès and Sperino with the matter of *soft* chancre mixed with vaccine lymph, an *irregular* vesicle resulted, which in its development had some considerable points of resemblance with a vaccine vesicle, but the matter from which, taken on the eighth day, produced only chancre (De la Syph. Vacc. Paris, 1865, p. 280). As regards true syphilis, however, one cannot see any reason why, if its inoculable products were mixed with vaccine lymph and inserted on the arm of a child, each infection should not within its own period of incubation produce its own specific results; and this is, in fact, by far the most probable explanation of one or two occurrences in which there seems authentic evidence of vaccinia and syphilis having been received at the same operation. (See Handbook of Vaccination, pp. 317–18.)

[2] See particularly the observations made as regards some of these by Simon, op. cit. p. lxvi.

Now the cases which have been brought forward, whatever ground they may give for caution (and in a matter of such extreme consequence there can never be too much caution), do not appear to me to afford the strict proof requisite, or indeed anything like it; on the contrary, each one of them is wanting in some essential point, or is open to some source of fallacy. Either there was no evidence that the child said to have originated the syphilis was at any time syphilitic; or it was not shown that the alleged syphilitic vaccination was not in fact a syphilitic inoculation *instead* of a vaccination; or there was a possibility, and indeed a probability, that the lymph used had been mixed with the inoculable products of syphilis; or there was reason to believe that the syphilis which developed itself after the Vaccination had an independent origin; or the facts were inquired into at too great a distance of time, and depended too much on the statements of ignorant persons to be wholly relied on. Thus, in the occurrences at Rivalta the circumstances were not inquired into till four months after their origin.

It is not intended in this article to consider the alleged cases of vaccino-syphilitic inoculation in detail. In none of them is the proof stronger than in the two to which I have already referred—the case of M. Trousseau at the Hôtel Dieu, and the syphilitic endemic at Rivalta: it will be sufficient, therefore, to examine these. In M. Trousseau's case a young woman was revaccinated from a child, healthy at the time, and, so far as was known, continuing healthy, from which child four children received their primary Vaccination, went through it perfectly, and (certainly) had no subsequent syphilitic affection. Only some small papules arose on the arm of the young woman, and the revaccination was considered to have failed. She remained in the hospital a month after the Vaccination, and *after being out another month* came back with two undoubtedly syphilitic sores on the arm on which she had been vaccinated. It was afterwards known, but not at the time, that she was a young woman of loose character. Now, assuredly no one who knows the extraordinary situations in which chancres have been met with, such as the cheek, the corner of the eye, all sorts of situations in which there was no suspicion of Vaccination, can say that there was no fallacy in this case, especially when we consider, first, that there was not a shadow of proof adduced that the child from whom the lymph was taken ever had syphilis, and there was every presumption that it had not; and secondly, the unusual length of the syphilitic incubation, supposing it dated from the time when the Vaccination was done.

The syphilitic endemic at Rivalta was a very remarkable occurrence, and under any point of view is of the deepest interest. Four other such endemics, said to have occurred in 1814, 1821, 1841, and 1856 respectively, have been ascribed also to a vaccinal origin; and not the least curious part of the matter is that all these should have occurred in Italy, and that none like them should have been met with in any other country. In their mode of communication, apart from the Vaccination—for many persons were affected who were not at the time the subjects of Vaccination—they bear a close resemblance to what we read of endemics of sibbens and other syphiloid diseases, recorded before Vaccination was known; not only was there communication of disease from babies to their nurses and from wives to their husbands, but children infected one another by the act of kissing, and we are even told that when poor people were crowded together in confined and neglected dwellings, whole families were affected. In the Rivalta endemic, the alleged vaccinal origin was made the subject of careful inquiry by a scientific commission. But unfortunately this inquiry did not take place till four months after the outbreak; and, as in no single case of the children said to have been syphilized by Vaccination had any application been made to a medical man on account of the condition of the arm, facts and dates, requiring the closest and most accurate observation, with careful record from day to day, had to be taken at that distance of time on the testimony of the parents and villagers. Under these circumstances we are not surprised that different conclusions were arrived at; and that though Dr. Pachiotti and the other commissioners, after a most careful and painstaking inquiry, reported themselves satisfied of the vaccinal origin of the disease, Sperino, who also went to Rivalta and saw the cases, and treated some of them afterwards at Turin, was equally satisfied that this origin was altogether independent of the Vaccination. The story, as regards the vaccinal origin, is this:—

A child (Chiabrera), apparently in good health, but really incubating syphilis, was vaccinated with some lymph obtained in a tube; this child's arm was used *on the tenth day* for the vaccination of 46 children, and one of these 46 children, named Manzone, *again on the tenth day* furnished lymph for vaccinating 17 children: of these 63 children 46 had, within two months, a disease considered by the commission to have been syphilis,—the syphilitic symptoms having manifested themselves in some cases *within ten days*, and *as a mean*, at twenty days from the Vaccination. It need scarcely be pointed out how irreconcilable these dates are with all that we have been taught as to the incubation period of constitutional syphilis. But supposing this teaching to have been erroneous (and, unless it is, the Rivalta cases as connected with Vaccination fall of themselves), and supposing that it is possible to produce the primary symptoms of constitutional syphilis within ten days of the inoculation of the poison, and that from Chiabrera's arm the whole mischief arose, there is still the question *from what sort of vesicle* on his arm was the lymph taken? We have not only the higher authority of Ricord that the chancrous pustule, "initial lesion of the primary syphilitic ulcer when it develops itself on the skin," has characters which may cause it to be confounded by careless persons with the vaccine pustule: but we know also that such a vesicle as may be produced by mixture of the matter of soft chancre with vaccine virus, *though according to the description given it would be impossible for any careful person to confound it with the regular vaccine vesicle*, has yet much of the vaccine character about it. Now of the vesicles on Chiabrera's arm we have no reliable account: nor of the vesicles on the arms of any of the other children, at the period of their so-called Vaccination. So far as we have details of the Vaccination, they are not such as to give us any confidence in the operation, and it is quite an open question whether the children were not carelessly syphilized instead of being vaccinated.[1] When the cases were seen by Dr. Catt, or by the Commission of Inquiry, they presented either so many syphilitic sores, or cicatrices which, according to the description given, had no vaccine character about them: and the revaccination of five of the children afterwards without effect (a revaccination which it is not stated was repeated) is far from being conclusive.

In the years which have passed since these occurrences became the subject of discussion, attention in this country has been wide awake to find any that should be like them. None have been met with. On one occasion one of our ablest, as he is one of our most candid workers in syphilis, met with a case which he stated to the Medico-Chirurgical Society was one of syphilitic infection communicated in vaccinating, but it turned out that even his experienced judgment had been deceived, and that, as he allowed subsequently, he had mistaken for a syphilitic sore a merely degenerated vaccine vesicle. Nor with all his vast experience of syphilis has he to this day met with any case in which there was ground for believing that that disease had been communicated in vaccinating.

The real lesson which, as it appears to me, is derivable from the alleged cases of vaccino-syphilitic inoculation, is the extreme care with which Vaccination should be conducted. We must not only be on our guard against any possible admixture of syphilitic matter with our vaccine lymph, either through the lancet or otherwise; but we must be careful that our lymph itself is taken only from the healthiest children, from the most perfect and regular vesicles at the proper period of their course, and that it is pure unmixed vaccine lymph, free from the slightest stain of blood. We have no right to run a *merely possible* risk that can be avoided.

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[1] Mr. Simon informs us that in a child having latent syphilis he has known a clean incision, made in performing a trifling surgical operation, develop in a few days a syphilitic sore. I do not say that Chiabrera's arm had such a sore on it; the state of his arm is the very thing regarding which we want evidence. Knowing the careless and miscellaneous way in which Vaccination has sometimes been carried on, and the ignorant hands into which its performance has sometimes fallen, I am rather surprised that accidents have not more frequently occurred. It is only a few years ago that I was the means of stopping a druggist, who was rather an extensive vaccinator, from proceeding to vaccinate some children from *an open sore* upon an arm which had nothing of vaccine character whatever about it, but which he assumed to be a vaccine sore, because he had vaccinated the child a week before: and the details of some of the Continental vaccinations in which accidents have occurred are truly astonishing. Further, the importance and necessity, in alleged cases of vaccino-syphilitic inoculation, of having precise details, and of using care and reserve in the admission of statements, are well illustrated by the particulars of a case often referred to in the discussions in the Académie to show that syphilis had been inplanted by a properly performed Vaccination. It is the case well known as that of "Le Vétérinaire B."—in which 19 out of 24 persons revaccinated by him had afterwards signs of syphilis, the stuff with which all were vaccinated being, it was said, vaccine taken "sur un enfant qui était fort, et qui paraissait complètement sain;" but concerning whom, further inquiry elicited this important information, "*On sut depuis* que l'éruption vaccinale ne s'était pas fait regulièrement chez lui, que le huitième jour il n'y avait pas encore trace de boutons!"

GLANDERS—EQUINIA.

By Arthur Gamgee, M.D., and John Gamgee.

Definition.—A febrile disease, due to the introduction into the system of a specific poison, originating in the horse, ass, or mule, and communicated directly or indirectly from them to man.

It is usually ushered in by rigors, which are followed by articular pains, and great prostration. There is more or less affection of the lymphatic vessels and glands, which inflame and suppurate. Ulcerations appear on the pituitary mucous membrane, from which there flows an aqueous or purulent discharge. A pustular eruption often occurs on the surface of the skin, which in parts becomes affected with inflammation of erysipelatous character. Abscesses form in the subcutaneous cellular tissue. The disease is usually fatal.

Nomenclature and History.—Under the name of Μήλις, Aristotle[1] described a disease affecting the ass, which was probably identical with the "malleus" or "morbus humidus" which an early writer on veterinary medicine, Vegetius, subsequently described as affecting the horse, and with the disease to which, from an early period, the name of Glanders has been applied by English writers. The term Glanders includes several affections, which, undoubtedly due to the same specific virus, must be looked upon as mere varieties of one disease, but which, differing very remarkably in character, were for a long period of time considered to be altogether distinct.

Vegetius, and the authors who followed him, described under the terms Malleus humidus, Morbus humidus,[2] Cymoira,[3] Cimoira,[4] Capitis morbus, that variety to which the term Glanders has been restricted by English writers—to an affection of horses which is characterised by ulcerations of the Schneiderian mucous membrane, accompanied by a discharge from its surface, and by enlargement and induration of the submaxillary glands; which may run a long or short course, and which may be, but often is not, accompanied by marked constitutional symptoms. They described in addition, under the terms Morbus farciminosus,[1] Vermis equi,[2] Vermis volativus,[3] Farcina equi,[4] Turtac, cutis equorum,[5] Glandulæ et scrophulæ equi,[6] an affection which has now been proved to be merely a variety of the one described, and of which the striking and characteristic feature is the formation of abscesses and swellings in the course of the lymphatic vessels and glands, and of small tumours beneath the skin, and which is unaccompanied by any affection of the Schneiderian membrane. This, which for a long period of time was looked upon as altogether a distinct disease from Glanders, received in, English, the name of Farcy, and will in the following pages be described as one of the important forms or varieties of Glanders.

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[1] Aristotle, De Hist. Anim. lib. viii. cap. xxv.
[2] Vegetii Renati Artis Veterinariæ, sive Mulomedicinæ, libri quatuor, &c. lib. i. c. vii.
[3] Laurentius Rusius, c. lxxi. p. 72, quoted by Heusinger.
[4] Jordanus Ruffus, c. xvi. p. 48, quoted by Heusinger.

As Glanders is a disease which always originates in the horse and ass, never occurring in man except when communicated, directly or indirectly from them, and the nomenclature of the disease in man having been borrowed, to a great extent, from that previously in use amongst veterinary writers, its complete history, in the first place, necessitates an account of the mode in which it originates in the lower animals, and of the forms which it presents in them.

GLANDERS AND ITS VARIETIES IN THE HORSE, ASS, AND MULE.—This disease appears to affect the horse in all parts of the world, although perhaps it is modified to a certain extent by climatic and other agencies. In the deserts of Arabia it is said not to possess the dreadful characters which distinguish it elsewhere, and is a comparatively rare disease. It may occur under four forms, as 1st, Chronic Glanders; 2d, Acute Glanders; 3d, Chronic Farcy; 4th, Acute Farcy.

CHRONIC GLANDERS is the most common form affecting the horse. It is propagated by contagion and infection (?). It never occurs as a termination of acute Glanders. Its period of incubation is uncertain, and has been stated to vary from a few days to a year (?).

SYNONYMS.—Morve chronique, French; Chronischer Rotz, German; Ciamorro cronico, Morva cronica, Italian.

SEMEIOLOGY.—The general health is little, if at all, affected. There is swelling and hardening of the submaxillary lymphatic glands. A discharge occurs from one or both nostrils, generally from one only (usually the left), which is at first of watery consistence, becoming more gluey, purulent, sanious, and fœtid. Elevations and ulcerations occur on the Schneiderian mucous membrane. A horse thus affected often appears to be in perfect health. When placed under unfavourable circumstances, especially if fed scantily, symptoms of acute Glanders rapidly make their appearance, and death then soon inevitably follows.

MORBID ANATOMY.—The mucous membrane lining the cavities of the nose and sinuses, presents small white elevations, varying in size from a small to a large pin's head, and larger patches of a yellowish-white colour, having a smooth surface. These elevations and patches soften in the centre, and then present the appearance of excavated ulcers. The ulcerations sometimes, though not usually, implicate the whole thickness of the mucous membrane, and affect the bones; perforation of the septum narium, and of the nasal bones, sometimes occurring.

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[1] Vegetius, op. cit. lib. i. c. iii.

[2] Jordanus Ruffus, c. i. p. 23.

[3] Ibid. c. ii. p. 27.

[4] Albertus Magnus, p. 92.

[5] Ibid. p. 589.

[6] De Crescentiis, lib. ix. c. xii. p. 275.

The mucous membrane of the larynx, trachea, and bronchi, presents at an early stage of the disease, little white elevations, resembling the cicatrices of leech bites; these afterwards coalesce, become injected, and ulcerate, giving rise to excavated ulcers, of a deep-red colour.[1]

The lungs contain small fibrinous deposits, varying in size from a pin's head to that of a grain of flax, around which the pulmonary texture presents quite a healthy appearance. Larger masses, of a bluish-white, lardaceous, or gelatiniform appearance, which sometimes are of the size of a hen's egg, are also observed. A condition of lobular pneumonia has been described to exist in certain cases. Although veterinarians have spoken of these alterations in the lung as tubercular, there is no ground for the opinion, as the researches of Rayer,[2] Tardieu,[3] and Trousseau[4] have proved.

Acute Glanders occurs more rarely in the horse than the chronic form, of which, as of acute and chronic Farcy, it is a frequent termination. In the ass and mule it is the common form of the disease.

Synonyms.—Morve aigue, French; Acuter Rotz, German; Ciamorro acuto, Morva acuta, Italian.

Like the chronic, the acute form of Glanders is an intensely contagious disease. The period of incubation is uncertain. It has been stated to be only from three to five days,[5] although it is undoubtedly longer in certain cases.

Semeiology.—The disease sets in suddenly, a short time (a few days) after exposure to contagion, with symptoms of inflammatory fever. The respirations are hurried. A copious yellow, purulent, or sanious discharge flows from the nostrils. There is watering of the eyes. Sometimes an eruption of small cutaneous, or subcutaneous, tumours occurs. Then supervenes violent inflammation of the pituitary membrane, which becomes deeply and extensively ulcerated. Cough and shortness of breathing occur. Death invariably follows.

Morbid Anatomy.—The mucous membrane, lining the nares and frontal sinuses, is found acutely inflamed, and generally presents a pustular eruption, which is bathed in a purulent fluid. If the disease lasts for some time, ulcers form, which are either small and round, or large and irregular; in the latter case having been formed by the ulceration of several confluent pustules. The ulcers are excavated, often appearing as if cut with a punch. The lungs are almost constantly the seat of limited pneumonia, and frequently purulent deposits are formed in them. Petechiæ occur on the surface of the pleura, pericardium, and peritoneum. In certain cases (morve aigue, hémorrhagique et gangréneuse, Rayer) petechiæ and large ecchymoses are noticed, after death, on the pituitary membrane. In these cases the mucous membrane is generally, over some part of its extent, destroyed, softened, and exhales a gangrenous odour.

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[1] Tardieu, De la Morve et du Farcin chroniques chez l'Homme et les Solipédes. Paris, 1843. P. 36.

[2] Rayer, De la Morve et du Farcin chez l'Homme. Mém. de l'Acad. R. de Méd. Tome sixième, pp. 828–833.

[3] Tardieu, op. cit. p. 41.

[4] Trousseau, Recherches Anat. et Pathol. faites à Montfaucon.

[5] Hering, Specielle Pathologie und Therapie für Thierärzte. Stuttgart, 1858. P. 98.

CHRONIC FARCY is, like the other forms of Glanders, highly contagious. It may be produced by the inoculation of the altered secretions of farcied or glandered horses.

SYNONYMS.—Farcin chronique, French; Ch. Hautwurm, Wurm, German; Farcino, Italian.

SEMEIOLOGY.—The disease usually commences by an indolent inflammation of the lymphatic vessels and glands, which become red, tender, and acquire a large side ; the swellings occurring chiefly in the situation of the valves of the lymphatics (farcy-buds).

An eruption of small subcutaneous and cutaneous tumours occurs. The large glands and cutaneous swellings have a tendency to suppurate, and indolent ulcers result from the opening of the abscesses ; these secrete an ichorous discharge, capable of producing Farcy, or Glanders, or both. The general health may continue good for a long period of time, the disease remaining stationary. If the animal be not destroyed, symptoms of general constitutional disturbance supervene. The animal loses flesh, has a staring coat, coughs, and usually falls a victim to acute or chronic Glanders.

MORBID ANATOMY.—Structural alterations of lungs and other organs, as in Glanders. Induration and swelling of the lymphatics.

ACUTE FARCY is distinguished from chronic Farcy by the rapidity of its course, the urgency of the constitutional symptoms, and by its being almost invariably associated with acute or chronic Glanders. The chief anatomical difference between the two forms is the occurrence, in the acute, of truly cutaneous abscesses or boils. Acute Farcy proves invariably fatal.

MORBID ANATOMY.—The same as that of Glanders and chronic Farcy.

Having described briefly the essential characters of the different varieties of Glanders, as they occur in the horse, ass, and mule, before proceeding to the consideration of the history of the disease as it affects man, it will be well to state shortly what appears to have been definitely made out, by veterinarians, as to the nature of the disease, the relations which exist between its various forms, and the mode in which it is transmitted. It has been satisfactorily proved,—

1st, That the different forms of Glanders are due to, or are associated with, the formation of a specific virus, which exists in the blood and secretions of animals affected with it. The virus is readily absorbed by an excoriated or wounded surface, or when matters containing it are injected into the blood. It is likewise probably absorbed by the unbroken surface of mucous membranes.

In one or other of its forms the disease may almost certainly be induced by introducing beneath the skin of a healthy animal, the nasal mucus of a glandered horse, or the purulent or ichorous discharge which flows from the ulcerated swellings of Farcy. It may be induced by injecting into the circulation of a healthy animal, the blood of one suffering from Glanders.[1] The mucous membrane of the alimentary canal appears to be capable of absorbing the virus under certain circumstances.[2] There is reason to believe, however, either that gastric digestion is capable of destroying the virus, or that it may pass through the alimentary canal without injurious consequences resulting, provided the mucous surface be not abraded.[3]

2d, The virus of glanders and farcy is identical. The same pus may, if introduced into the system of one horse, produce acute glanders; into that of a second, farcy; into that of a third, chronic glanders.

On certain points the opinions of veterinary writers have been much divided. The chief subjects of dispute are, (1) the possibility of the disease originating spontaneously; (2) the mode of its transmission; whether always by contagion, or by contagion and infection.

The intensely contagious nature of the disease is admitted at the present day by all writers; nearly all, however, admitting the possibility of the disease originating spontaneously. An impure and confined atmosphere, excessive work, and insufficient food, are the chief causes which have been alleged to induce it. That these circumstances favour its spread, cannot be doubted; that they act as most powerful predisposing causes, to this as to other diseases, has been unequivocally proved, by the ravages which Glanders has so often caused amongst the horses of armies subjected to famine and other injurious influences. French writers have all very strenuously maintained the possibility of the spontaneous origin of Glanders, a position which was strongly held by our countrymen, Professor Coleman, who certainly has been one of the chief contributors to our knowledge of this disease. It was alleged, in an important discussion on this matter in the French Academy of Medicine, in 1861,[4] that at a period when the fortifications of Paris were being constructed, the horses of the poor and small contractors were found to suffer to a great extent from Glanders, whilst those belonging to the wealthier contractors escaped the disease. Those who adduced these facts attributed the occurrence of the disease, in the former case, to the insufficient nourishment and the harder work which the animals belonging to the poorer contractors received, whilst they appear to be susceptible of a very different and much more likely explanation. The laws which exist in France for preventing the use of glandered horses have been inefficiently carried into execution, glandered horses being by some people systematically employed; these horses are necessarily very much cheaper than sound ones, and would be more likely to be bought by the poor, than by the rich contractor. In our own country Glanders was, at one time, a most prevalent disease, which created the greatest ravages. Glandered horses were systematically sold and worked. The practice caused the greatest pecuniary loss to the country at large, for one case of Glanders having been introduced into a stable, the chances were considerable that a majority of the horses in it would sooner or later die of the disease. To remedy this state of matters a most stringent law was passed, which prevented the working of glandered horses. Carried into execution, with great rigour, this law has had the effect of rendering Glanders, in all its forms, a very rare disease in this country. Thousands of horses are exposed to those causes which are supposed to give rise to Glanders; are hard worked, ill fed, and kept in badly-ventilated stables, in the same way as horses used to be a century ago, but yet Glanders does not originate. Glanders has now almost ceased to affect the horses of our army, and the sporadic cases which very rarely occur need cause no astonishment when we remember that the disease is one which still prevails to a certain extent, especially in Ireland, whence most of our cavalry horses are obtained, and that the disease may have a long period of incubation. The history of Glanders amongst the lower animals in this country leads us to form the opinion that it never originates spontaneously in our climate. We must fully admit, however, as we have already done, that bad feeding, bad ventilation, and excessive work are powerful predisposing causes of this disease, and to a great extent explain the ravages which it has often caused amongst the horses of armies, which in time of war, from the large and indiscriminate purchases of horses that must necessarily be made, contain abundantly the germs of the disease.

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[1] Travers, An Inquiry concerning that disturbed State of the Vital Functions usually denominated Constitutional Irritation. London, 1826. Vol. i. p. 355.

[2] Sainbel, Vial de St., Experiments and Observations on Glandered Horses, p. 109.

[3] Parent Duchâtelet, Hygiène Publique, tome xi. p. 194.

[4] Recueil de Médecine Vétér. Août 1861, p. 645.

Whether actual contact of some article, containing the virus, with a mucous membrane, or a broken cutaneous surface, be required to induce the disease, or whether its germs may be communicated through the atmosphere—whether, in short, Glanders is always communicated by contagion, using the term in its more limited sense, or by contagion and infection, has been warmly argued by various writers. Probably in nearly every case actual contact of glandered matter with an absorbing surface does take place. The mucus flowing from the nose of the glandered horse becomes attached to the stable and the stable utensils, and comes almost necessarily in contact with the water, hay, and straw, which horses in the same stable employ, so that they are frequently exposed to conditions *positively* known to be capable of inducing the disease.

Some cases have, however, been recorded which cannot well be explained on the hypothesis of actual contact, and we therefore do not deny the *possibility* of Glanders being transmitted from one lower animal to another through the medium of the atmosphere, although we believe this to occur, if at all, only very rarely.

History of the Disease in Man.—No connexion had been traced between the terrible diseases in the lower animals which have been briefly described, and an affection which then as now must have occasionally affected those who had charge of horses suffering from Glanders and Farcy, until the year 1810, when Waldinger[1] drew attention to the fact that special precautions ought to be adopted in the dissection of horses affected with Glanders and Farcy, inasmuch as the direst consequences, even death, might result from the inoculation of the purulent matter. The accuracy of the statements of Waldinger was supported by the publication, in 1812, of a paper by a French military surgeon, Lorin, who, under the title "Observations sur la Communication du Farcin aux Hommes," described the case of a veterinary surgeon who, having accidentally pricked himself whilst operating upon a glandered horse, suffered in consequence from inflammation of the hand.[2] The statements of Waldinger do not, however, entitle him to be considered the first person who pointed out that Glanders is communicable from the lower animals to man; for although he stated that dangerous consequences might result from the inoculation of the purulent matter of Glanders and Farcy, he did not state that the affection induced in man in any way resembled that of the horse. Again, the observation of Lorin was of the most unsatisfactory description, for the case which he describes does not differ materially from many cases of dissection wounds, and possessed, in no respects, the peculiarities of Farcy. Schilling, however, published in 1821,[3] under the title "Merkwürdige Krankheit und Sections Geschichte einer wahrscheinlich durch uebertragung eines thierischen Giftes erzeugten Brandrose," the case of a man who, having washed out the nares of a glandered horse, became affected with a pustular eruption on the skin, an offensive discharge from the nostrils, and erysipelatous inflammation of the face, and who died after an illness of eight days. Rust looked upon this as a case of Glanders in man, and in support of this view gave the notes of another case of a similar nature. Both these were indeed most typical cases, and must be considered to be the first well-marked cases of Glanders occurring in man which were published. In the same year that Schilling's and Rust's cases were published, there appeared in the *Edinburgh Medical Journal*[4] a short notice copied from a subscription paper in the Hotel Coffee-house, Leeds. This short notice (of nine lines) was signed by T. Muscroft, surgeon, Pontefract, and consisted in an appeal on behalf of the family of the dog-feeder of the hounds belonging to the Badsworth Hunt, who, in cutting up the carcase of a horse which had died of Glanders, had accidentally wounded his hand. "In a few days he betrayed all the symptoms which are at first shown in the horses beginning in the above disease. He gradually became worse, and at the end of the week he died raving mad, labouring under a confirmed complaint of Glanders."

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[1] Waldinger, Wahrnehmungen in Pferden. 2te Aufl. Wien, 1810.
[2] Journ. de Méd. Chir. et Pharm., Fév. 1812.
[3] Rust's Magazine, vol. i. p. 480.
[4] Edin. Med. and Surg. Journ. vol. xviii. p. 321.

Feeling considerably interested in reading this notice, I wrote to Henry Muscroft, Esq. surgeon, of Pontefract, asking if the notice which had appeared in the Edinburgh Journal in 1821 had been written by a relative of his, and requesting to be put in possession of any information he might possess on the matter. Mr. Muscroft informed me in reply that the notice had been signed by his father, who at that time was practising in Pontefract, and added that he had never heard anything about the case. Stimulated by my questions, Mr. Muscroft, however, instituted inquiries, which ended in his discovery of the widow of the unfortunate man. The old woman is now eighty-six years of age; she was, however, able to give a very complete account of her husband's illness. This account was written down verbatim by Mr. Muscroft, who has sent it to me. Information obtained from other sources proves that the dates mentioned in this statement are perfectly correct. Mr. Muscroft's pay-book, on being referred to, shows that he attended John Turpin from March 18, 1821, to March 27; and the parish register of deaths contains an entry to the effect that John Turpin died on the 29th March. The statement of the old woman is interesting, as referring to the first case of Glanders, which appears to have been recognised as such, in this country.

Statement of Sarah Hazelgrave (formerly Turpin), aged eighty-six years.

My first husband, John Turpin, whilst skinning a dead horse, in the month of March 1821, cut the third finger of the left hand, and, heeding but little this accident, on the following day he left me for a week, to fetch home to the Badsworth kennels some young hounds that had been reared in the neighbourhood of York.

On his arrival at home, he was very weak and scarcely able to walk, and he at once said that "he should never go out again, he was so ill." He complained of severe pain in the head, and there was profuse discharge from the nostrils; and on different parts of his body there were a number of blisters of different sizes, which, after a time, became blue.

Mr. Muscroft was sent for, and after he had been at my husband's bed-side for some time, he said, he was "afraid Turpin was inoculated by the blood of the dead horse he had cut up, and that there was no remedy, the disease being horse-farcy, or glanders, but that, if the finger had been taken off at first, this might not have happened."

Mr. Muscroft gave him medicines, and saw him daily until his death, which took place ten days after he returned from York. He retained consciousness to the last. Large lumps appeared on his forehead and face and his throat was swelled. His head and face became very large. The wound of his finger was very bad, and the finger looked as if it would rot off, and from the wound there were hard cords, like the stem of a pipe, up the arm, and the armpit was also swelled. The smell was very bad.

May 18, 1865.

Before the publication of the cases of Schilling and Rust, and before the notice referred to appeared in the *Edinburgh Medical Journal*, certain cases of transmission of Glanders from horses to man had been observed in London, which, although not published until some years later, received at the time a thorough investigation—a study which first and satisfactorily proved that the virus of Glanders is transmissible from horses to man, and generates in him affections which, although slightly different in some of their characters, are identical with the disease as it is observed in the horse. These observations were first published by Mr. Travers, at page 350 of the first volume of his work on "constitutional irritation."[1] The first and most interesting of these cases occurred in the year 1817, and was that of Mr. William Turner, a veterinary student, who injured his finger in examining the head of a horse which had died of Glanders. An ulcer followed, with inflammation of the absorbents and cellular tissue of the hand, and symptomatic fever. After some days an abscess formed in the opposite arm, and another in the lower part of the back. Matter taken from the abscess of the arm was sent to Mr. Coleman, who inoculated an ass with it, and produced fatal Glanders. Mr. Turner's health was seriously affected. He became hectic, and was sent to Brighton for the benefit of sailing and tepid bathing. Subsequently an abscess formed in his lungs, another in the kidney, and after his return to his residence at Croydon, at the expiration of several months, abscesses formed successively upon each knee-joint. From the last, matter was taken for the purpose of inoculating an ass, which in eleven days died glandered. Shortly after this, Mr. Turner's long and painful illness terminated in death. The third case, recorded by Mr. Travers, that of Nimrod Lambert, is however of greatest importance. N. L., a healthy hackney coachman, æt. 32, in January 1822, infected a chap on the inside of the right thumb, by inserting it into the nostril of a glandered horse, to pull off a scab. He was taken suddenly ill three days after, and suffered subsequently from inflammation of the lymphatic vessels and glands of the arm, which suppurated and ulcerated. "The glands at either angle of the lower jaw, and in the groin, became swollen, and he was much afflicted with pain between the eyes and down the nose, and exulcerations of the membrana narium, attended with discharge." After a long and painful illness the man recovered, although his constitution appears to have been violently affected and permanently injured by it. An ass, inoculated with the matter from this man's sores by Mr. Sewell, died of Glanders. The great interest attaching to Lambert's case rests in the fact that it is the first *well-marked* case of Glanders occurring in man in which the diagnosis was rendered perfectly certain by the inoculation of animals with the purulent matter. Mr. Turner's case, on the other hand, was by no means a well-marked instance of chronic Farcy, but is valuable as being the first case which proved that in its passage through the human body, the virus of Glanders loses none of its properties, and is again able to generate the disease in lower animals. With these cases Mr. Travers published a letter from Professor Coleman, in which that gentleman described the experiments which he had made with the pus obtained from Mr. Turner's abscess. In this letter Mr. Coleman stated the result of a most interesting experiment, in which he succeeded in inducing Glanders, "by removing the healthy blood from an ass, until the animal was nearly exhausted, and then transferring from a glandered horse blood from the carotid artery into the jugular vein." (Op. cit. page 355.) Although Mr. Travers published these cases, he does not appear to have considered the disease induced in Mr. Turner and Lambert, to have been *glanders.* "The reader," says Mr. Travers, "cannot fail to perceive some points of analogy between the effects produced by the absorption of glandered matter, and of the fluids of the human body after death. I think there can be no difficulty in admitting in either of these cases that a poison was imbibed, but the evidence gives no ground for the conclusion that it was a morbid poison, as regards its operation in the human subject." (Op. cit. p. 365.)

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[1] An Inquiry concerning that disordered State of the Vital Functions, usually denominated Constitutional Irritation, by Benj. Travers. London, 1826.

Grubb,[1] Krieg,[2] and Brown[3] described after this several interesting cases of Glanders transmitted from the horse to man. It was, however, by the publication of a memoir entitled "On the Glanders in the Human Subject,"[4] that Dr. Elliotson attracted the attention of the profession to this subject. In this paper he gave the history of two interesting cases of Acute Glanders, which had been observed by Dr. Roots and himself, and of a third which had been communicated to him by Mr. Parrott, of Clapham, and he collected nearly all the observations which had, up to that date, been placed on record. In a second memoir,[5] entitled "Additional Facts respecting Glanders in the Human Subject," Dr. Elliotson gave an account of another case of acute Glanders which had fallen under his notice. Subsequently to the date of Dr. Elliotson's papers, several cases of Glanders were published in various journals. No very valuable addition to the knowledge of the subject was however made until the year 1837, when Rayer, in a splendid memoir, entitled "De la Morve et du Farcin chez l'Homme,"[6] gave an account of all the cases of Glanders which had been observed up to that date, and gave an original and complete description of the different forms of Glanders both in the

[1] Diss. sistens casum singularem morbi contagio mallei humidi in hominem translato orti. Berolin, 1829.

[2] De Typho malloide. Berolin, 1829.

[3] London Medical Gazette, vol. iv. p. 134.

[4] Med.-Chir. Trans. vol. xvi. 1830.

[5] Ibid. vol. xviii. 1833.

[6] Mém. de l'Académie de Médecine, vol. vi. p. 625.

horse and in man. The subject of Chronic Glanders and Farcy was afterwards made the subject of special investigation by Tardieu, who, in a memoir entitled "De la Morve et du Farcin chroniques," supplemented the knowledge which Rayer had already collected on these subjects. It is to the researches of these distinguished physicians that we owe almost the whole of our knowledge of the morbid anatomy of the various forms of Glanders.

Since the date of the publication of these memoirs, many cases of Glanders possessed of great interest have been recorded: no very important addition to the existing knowledge of the disease has, however, been made.

ETIOLOGY OF THE DISEASE IN MAN.—This section of our subject has been already so fully treated of in discussing the subject of Glanders affecting the horse, that it here needs but a very brief consideration.

Always communicated from the horse, the ass, or the mule to man, those are specially predisposed to contract the disease whose avocations lead them to come in contact with these animals. The great majority of the cases recorded have occurred amongst veterinary surgeons, veterinary students, grooms, coachmen, cavalry soldiers, horse-slaughterers, &c. Writers on Glanders in man have admitted that, although the disease is often or generally due to inoculation of morbid matters from glandered horses, it may be occasionally communicated to man by infection. It has been stated, in support of this view, that a frequent cause of Glanders and Farcy is the custom of causing men to sleep in stables. Those who hold the above view seem to forget that these men, besides sleeping in the stables, are, from the very nature of their duties, exposed to the greatest danger of contracting, *by inoculation*, Glanders from any affected horse which may be placed under their charge. If facts are very scanty to support the belief that Glanders is propagated from horse to horse by infection, they assuredly are still more so in the case of man. In the immense majority of cases of Glanders in man which have occurred, there is the most conclusive evidence that the sufferers were in constant habit of *handling* glandered horses, whilst in a considerable number of cases there has been actual evidence of the disease having been transmitted by inoculation. To explain, as due to infection (using the term in its more limited sense), the cases which have occurred in which no history of inoculation through a wound is to be obtained, appears to the writer altogether unjustifiable, when regard is had to the facts that the virus of Glanders, besides being capable of being absorbed by the most trifling cutaneous abrasion, is probably absorbed by unbroken mucous membrane, and that of the very large number of persons who formerly used to be brought in contact with glandered horses, only an excessively small fraction contracted the disease, although a large number of these people were most careless and imprudent in their treatment of glan-

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[1] Paris, 1843.

dered horses. It may, we think, be safely stated that Glanders is only communicable from the lower animals to man by inoculation.

The virus of Glanders may adhere to stable utensils, clothes, and other articles, for a long period of time, and then when brought in contact with an absorbing surface may give rise to the disease.

Not only can the disease be transmitted from the horse, ass, or mule, to man, but likewise from one human being to another. Several facts have placed this beyond dispute. A distinguished young French veterinarian, M. Gerard, died of Acute Glanders induced by a wound which he inflicted on himself whilst making the post-mortem examination of a veterinary student who had died of Glanders.[1] Elliotson,[2] in describing a case of Glanders, mentions that the laundress who washed the clothes of the patient, contracted Glanders. An externe of a Paris hospital died from Acute Glanders contracted from a stableman suffering from Glanders.[3] In this case, besides dressing the wounds of the patient, the externe had taken an active part in the examination of the body after death.

Although usually contact of the virus of Glanders with an absorbing surface gives rise to the disease, this does not invariably follow.

It has been erroneously stated that a weak state of the constitution, intemperate and irregular habits, predispose individuals to attacks of Glanders. Whatever influence they may have on its progress and issue, there is absolutely no proof that they exert any influence in the way mentioned. It has, indeed, been correctly remarked, that a large majority of the recorded cases of Glanders have occurred in men of robust constitutions, and in the prime of life.

SEMEIOLOGY.—We shall consider separately the semeiology of the four varieties of Glanders which are observed in man as in the horse.

1. *Acute Glanders* occurs in man more frequently than the other forms of the disease, and presents in him characters resembling those of cases in the horse in which Acute Glanders and Farcy are combined. The period of incubation of Acute Glanders varies probably from twenty-four hours to a fortnight; its limits, in the majority of cases, being from three to eight days.

The mode of invasion of the disease varies considerably. In those cases where a distinct wound exists, through which the virus has been introduced, redness, pain, and tension, accompanied by an erysipelatous appearance around the wound, and swelling of the lymphatics, are often noticed before the supervention of any constitutional symptoms. Occasionally no wound can be discovered, or the wound through which the virus was introduced may remain in a passive condition. Whether local symptoms have been developed or not, the disease is usually ushered in by feelings of lassitude, headache, and rigors, frequency of the pulse, and often by vomiting and diarrhœa. Articular and muscular pains occur from an early period of the disease, and increase during its progress. The limbs and body become the seat of subcutaneous abscesses, which are specially found on the face, and near the articulations. Over the abscesses the skin becomes of a red and violet colour, and sometimes limited gangrene sets in. The pus which forms in the abscesses is serous and fœtid. A remarkable pustular eruption generally appears on the surface of the body, being specially found on the cheeks, arms, and thighs. The pustules commence as little red spots, which afterwards present the appearance of white papules, and become full of pus; they are often surrounded by a red areola. The pustules vary greatly in size, presenting a flattened or pointed surface, and either occur singly or so closely aggregated as to be almost confluent. The pustules are often accompanied by bullæ of a dark colour, on the face, trunk, and organs of generation. An erysipelatous inflammation of the nose, eyes, and neighbouring parts of the face, sometimes extending to the scalp, is far from infrequent. The cutaneous eruption, which has been described, has been compared to that of varicella, variola, ecthyma, and yaws, but is considered by all the best writers on Glanders to be quite distinct and characteristic. (Rayer.) Amongst the most constant and pathognomonic of all the symptoms of Glanders, is however one which has been noticed in the large majority of the recorded cases. A yellow, viscous, purulent discharge, often mixed with blood, and not infrequently possessed of great fœtor, exudes from the nares. This proceeds from the Schneiderian membrane, which is *invariably* the seat of a pustular eruption, or of ulcerations.

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[1] Breschet, Revue Méd. tome ii. p. 96, 1826.

[2] Elliotson, Lancet, 1838.

[3] Tardieu, op. cit. pp. 140, 141; and Bérard, Bull. de l'Acad. de Méd. Nov. 1841, tome vii. p. 182.

The mucous membrane of the mouth is sometimes likewise the seat of ulcerations or pustules, and from it, in certain cases, a purulent fluid has been observed to exude. The submaxillary lymphatic glands are usually not enlarged.

The prostration, which has been stated to usher in the disease, increases during its progress. The pulse becomes excessively frequent, small, and compressible. The voice is feeble, and the breath fœtid. The constipation, which frequently exists at the commencement of the disease, is replaced by diarrhœa; the stools are extremely fœtid. A hard, dry cough, accompanied by a little expectoration, and often by difficulty of breathing, testify to the existence in man, as in the horse, of pulmonary complications. With the increasing prostration, delirium sets in, which is followed by coma, and ultimately by death. The duration of the disease has varied from three to fifty-nine days. As a rule, death has occurred about the end of the second or commencement of the third week. Acute Glanders runs an unusually rapid course when it occurs as a sequela of the other and more chronic forms of the disease.

Chronic Glanders is the rarest form of the disease in man. When it occurs, it is usually as a sequela of Farcy. Uncomplicated by Farcy, the disease has but seldom been observed. When Chronic Glanders occurs primarily (*i.e.* not as a sequela of Farcy), it commences with lassitude and very severe articular pains, which readily pass away, and are followed by a cough, sore throat, and a disagreeable feeling in the nose.

When Farcy has existed before the appearance of Chronic Glanders, no lassitude or pains may be noticed. In these cases, the first symptoms are the sore throat, cough, and affection of the nose, which have been alluded to. A pain is then felt in the trachea, the voice becomes altered in character, or true aphonia may occur. The cough is accompanied by dyspnœa, and often by expectoration. Capillary bronchitis and pneumonia sometimes supervene. The disagreeable sensations in the nose increase—the patient complaining of a feeling of its being stopped up; sometimes, although by no means always, there is a dull aching pain at the root of the nose. On blowing the nose, a puriform mucus, mixed with little bloodclots, becomes detached, or there may be a regular discharge (*jetage*) from the nose. On examining the nares at this stage, ulcerations may not infrequently be perceived on the Schneiderian membrane. By the introduction of a probe, inequalities of the surface, and even perforation, of the septum narium, may be detected.

Ulcerations may sometimes be likewise observed in the pharynx. There is very rarely any induration of the submaxillary glands.

The skin is free from eruption.

In addition to the special phenomena which have been described, the patient is usually prostrated, suffers from articular and muscular pains, diarrhœa, and nausea, loses flesh, and falls into a state of marasmus. He may die of simple exhaustion, or Acute Glanders may set in and rapidly close the scene. This form of Glanders has been once observed to terminate in recovery (case of Nimrod Lambert). Its duration is excessively various; those cases which are complicated with Farcy proving more rapidly fatal than others. As a general rule, the course of the disease extends over several months, and cases have been recorded where it appears to have lasted for some years.

Acute Farcy presents, almost constantly, all the characters which have been described as those of Acute Glanders, with the single exception that there is a total absence of affection of, or discharge from, the nares. So great is the resemblance between the two affections, that only certain special points in connexion with Acute Farcy need be alluded to. A subdivision of all cases of Acute Farcy may be made, into cases accompanied by a cutaneous eruption, and cases unaccompanied by eruption. In some cases of Acute Farcy, there is merely an inflammation of the lymphatic vessels and glands, accompanied with the formation of soft subcutaneous tumours in various parts of the body. In other, and by far the greater number of cases, an eruption, exactly similar to that of Acute Glanders, occurs, such cases being distinguished from instances of the latter disease only by the absence of affection of the nose. These cases are very much more serious than those without eruption, and follow exactly the same course as Acute Glanders, proving fatal usually between the thirteenth and nineteenth day. (Rayer, op. cit. p. 787.) In the cases where there is no eruption, and which have received the special name of cases of acute farcinous angeioleucitis, the disease very frequently terminates in recovery, or passes into Chronic Farcy.

Chronic Farcy may exist alone, or associated with Chronic Glanders. It is ushered in by lassitude, wandering pains, uneasiness, anorexia, accompanied by fever, which often assumes a tertian type. An abscess usually forms on the forehead, the calves of the legs, or some other part of the body, which is followed by other indolent and fluctuating tumours. Some of these open spontaneously, and give exit to blood, or ill-conditioned pus. The lymphatic glands only secondarily, and to a slight extent, become affected. With the successive appearance of abscesses, the general health and strength of the patient decline; he loses flesh; the skin becomes dry, and earthy; the countenance yellow and livid. Frequent rigors occur. The open abscesses become converted into horrible ulcers, which have no tendency to heal. A dry cough harasses the patient, who, with his body covered with foul ulcers, falls into a condition of most miserable marasmus. The course of the disease is tedious and uncertain. The abscesses commence to form from the third to the fifteenth day of the disease. The subsequent stage is, however, most protracted and uncertain in its issue. It may terminate by an attack of Acute Glanders, or of Chronic Glanders, or the patient may die of exhaustion, or from an attack of pyæmia. Lastly, recovery may occur. The duration of the recorded cases of Chronic Farcy has varied from four months to three years; it usually terminates in from ten to fifteen months. Of twenty-two cases of the disease, cited by Tardieu, six recovered.[1]

DIAGNOSIS.—Having described the characters of the different varieties of Glanders, there remains for consideration their differential diagnosis.

Acute Glanders is distinguished by such remarkable characters, which are always associated together, that when fully developed it would be impossible to confound it with any known disease. In its early stage, when the articular and muscular pains are very urgent, it might be mistaken for acute rheumatism. The occupation of the patient, the existence of a wound in a state of irritation, the much greater degree of prostration than is common in acute rheumatism, the absence (usually) of redness and swelling around the painful joints, would arouse the suspicions of the physician. At a later stage, the association of the peculiar and characteristic eruption of pustules and bullæ, with ulceration of, and discharge from the nose, taken in connexion with the history of the affection, distinguish in the clearest manner Acute Glanders from erysipelas of the face, typhoid fever, malignant pustule, pyæmia, and some other diseases, to which it has been supposed to present points of resemblance.

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[1] See Tardieu, op. cit. p. 75, from whom the above description of Chronic Farcy has been abridged.

Chronic Glanders offers much greater difficulties in diagnosis than the acute form of the disease. When uncomplicated by Farcy, it might be readily mistaken at first sight for one of the different forms of ozæna. The occupation of the patient, the mode of invasion of the affection, the absence of other evidences of strumous or syphilitic affections, would furnish the data for forming a diagnosis. Cases of uncomplicated Chronic Glanders are, however, as has been already stated, of great variety. In the cases where Chronic Glanders and Farcy are combined, the diagnosis is more simple, for an association of symptoms then exists which is not presented by any other disease. Cases of Chronic Glanders must, however, occasionally of necessity occur, in which the physician must experience great difficulty in forming a correct diagnosis. In these cases an ass, or a horse, might be inoculated with some of the nasal mucus, if the case were one of uncomplicated Chronic Glanders; or with pus from an abscess, if the affection were Chronic Glanders and Farcy.

Acute Farcy, when occurring in a characteristic form, could scarcely be mistaken for any other disease. The history of the case, the great rapidity of its course, and the extraordinary pustular eruption, together with the numerous subcutaneous abscesses and tumours, would distinguish the case from cases where multiple abscesses occur from the introduction of other septic matters into the system. In cases of Acute Farcy without eruption, and of uncomplicated Chronic Farcy, only an accurate acquaintance with the early history of the case, and an accurate study of its progress, could enable the physician to arrive at a correct opinion. The supervention of Acute or Chronic Glanders would, in a large proportion of such cases, remove the difficulties which they at first present.

Morbid Anatomy.—Having described, in the preceding pages, the morbid anatomy of the various forms of Glanders in the horse, there remains but little to be said of the morbid anatomy of the disease as it affects man, for the structural alterations of the nasal fossæ, of the larynx, trachea, and lungs, are as characteristic of the disease in man as they are of it in the horse. The chief point of difference in the morbid anatomy appears to be that, in all the different forms of Glanders seen in the horse, the lymphatic system is very much more involved than in man.

1. *Acute Glanders.*—In all cases where the nasal fossæ have been examined, they have been found the seat of disease. Sometimes the Schneiderian membrane is ecchymosed and gangrenous, whilst often it presents numerous little pustules of the size of millet seeds. The frontal sinuses are frequently found filled with puriform mucus. The larynx has been the seat of eruption and ulceration in several cases. The lungs frequently present patches of pneumonia, or purulent formations are formed in them.

2. In *Chronic Glanders*, the affection of the nose differs considerably in character. At an early stage, the mucous membrane is often

found injected and much swollen. Small submucous abscesses form. These open and become ulcerated; the ulceration spreads to the bones and cartilages, and necrosis generally follows. These ulcerations generally occur on the septum, which, in almost every case of Chronic Glanders, becomes ultimately perforated. Ulcerations of the larynx, trachea, and bronchi are very commonly found. The epiglottis is sometimes ulcerated, but the vocal cords are usually unaffected. The ulcerations of the trachea are often very extensive, and affect the whole thickness of the mucous membrane. They exhibit a remarkable tendency to heal spontaneously. The lungs are, by no means so constantly as in Acute Glanders, the seat of disease. The pleura is often covered with small yellow elevations, which, on being incised, are found full of pus. Beneath the pleura, deposits of a purely fibrinous nature are found. Occasionally a portion of lung substance is indurated, in consequence of fibrinous exudation; in the centre of the indurated portion, pus is formed. The lymphatic glands are often somewhat enlarged, reddish, and of soft consistency. The bronchial glands have, in cases where ulcerations of the air-passages existed, been found enlarged, softened, and in a state of suppuration. The alimentary canal, the liver, and spleen present no peculiar appearance.

The morbid anatomy of Acute Farcy is identical with that of Acute Glanders, except that the Schneiderian membrane is found perfectly healthy.

In Chronic Farcy, the morbid appearances resemble those of Chronic Glanders. There is, however, no affection of the nose.

Prognosis.—In all the forms of Glanders, the prognosis is of the most unfavourable description. Acute Glanders and Acute Farcy, when accompanied by the characteristic eruption, are almost necessarily fatal. One case of Acute Glanders, and several of Acute Farcy, have been recorded, in which recovery took place. In Farcy, unaccompanied by eruption, the prognosis is much more favourable, recovery being the rule, and not the exception. Chronic Glanders, especially when complicated by Farcy, is almost invariably fatal. The case of Lambert, which has been referred to, proves, however, that even under these desperate circumstances recovery may take place.

Chronic Farcy, although a most dangerous disease, usually terminates in recovery.

In forming a prognosis, in apparently slight cases of Chronic Glanders or Farcy, it must not be forgotten that Acute Glanders often suddenly supervenes, in the course of these affections, and then invariably proves fatal.

Therapeutics.—This section of the subject need unfortunately be treated of very briefly, for no treatment which has been adopted, in the cases which have hitherto occurred, has appeared to exercise the slightest influence in checking or modifying the progress of the disease.

Characterised as it is by symptoms of the greatest constitutional prostration, a stimulant and supporting plan of treatment appears to be indicated, and has been recommended, by all writers on Glanders.

All attempts which have been made to cure Glanders in the horse have proved futile. There is considerable evidence, however, that arsenic, especially when administered with nux vomica or strychnia, has in some cases of Chronic Glanders excited a remarkable influence in checking the progress of the local affections; and we therefore think a trial of these remedies might with propriety be made, in cases of Chronic Glanders occurring in man.

HYDROPHOBIA.

BY JOHN GAMGEE, AND ARTHUR GAMGEE, M.D.

DEFINITION.—A disease due to a specific animal poison which resides in the saliva of animals affected with it. It never originates spontaneously, and is communicated directly or indirectly, and usually by biting, from carnivorous or omnivorous animals, and especially dogs, to man. It is alone recognised by its physiological effects on man or animals. It induces pain and stiffness in the inoculated part, exalted sensitiveness and irritability, feverishness, mental anxiety, flitting pains, spasm of the throat on the sight of liquids, tremors, headache and delirium, vomiting, eructations and tympanites, great prostration and death. Its period of incubation varies from a few days to many months.

SYNONYMS.—*λύσσα, κυνόλυσσα, ὑδροφοβία*; Phobodypson, Pheugydron, Rabies, Rabies canina, Rabies contagiosa, Entasia Lyssa, Canine Madness; La Rage, Hydrophobie (French); Die Hundswuth, Wasserscheu, Tollwuth (German); Rabbia, Idrofobia (Italian); Hidrophobia (Spanish); Watervrees (Dutch).

HISTORY.—Few diseases have, at different periods of the history of medicine, excited greater attention, curiosity, and study than Hydrophobia, and few have been described in a more graphic or more accurate manner by ancient writers. Celsus,[1] Dioscorides,[2] Cælius Aurelianus,[3] and Galen, have left us accounts of the disease which equal those of the best writers who have followed them.

It has been argued, and indeed generally admitted, that the disease was not unknown to Homer, and the word *λύσσα*, which is so often employed in the Iliad to describe a wild and reckless fury, is supposed primarily to have been used to designate the madness of dogs.

This view has been held since the days of Cælius Aurelianus, and is supported by the best authorities.[1] Thus Dr. Bardsley, in a very learned memoir on Hydrophobia,[2] says :—

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[1] A. C. Celsi Med. lib. v. cap. xxvii.

[2] P. Dioscoridis Op. omnia. Vienna edition, 1598. *Σημεῖα κυνὸς λυσσῶντος, ι α π᾽ αὐτοῦ δεδηγμένων. κεφ. α.*

[3] C. Aurelianus, De Morbis acutis et chronicis, lib. iii. cap. ix.—xvi.

"We have already said that the disease was well known to Homer, and applied by him, with his usual critical exactness of similitude, to the indiscriminate havoc with which Hector sweeps through the battle-field of his enemies;" and after quoting several passages[3] in which the word *λύσσα* or one of its derivatives is used, he adds: "The poet with much propriety puts these words into the mouth of Ajax his enemy, for dog was already a term of reproach among the Greeks as well as the Jews." The passages alluded to do not appear to us to prove satisfactorily that their author was acquainted with Hydrophobia. The word *λύσσα*, and its derivatives, is used by Homer on all occasions, as it is by many other Greek writers,[4] to denote martial rage; and in spite of the occurrence of the expression *κύνα λυσσητῆρα*, we feel inclined to believe that this was the primary meaning of the word, which was only secondarily applied to designate the disease of the dog, in the same manner as madness, rage (Fr.), and rabbia have in modern European languages been secondarily used in a limited and specific sense to denote Hydrophobia. Excepting that the word was subsequently used to designate the madness of dogs, there is no evidence that it had that meaning at the period when the Homeric poems were written; whilst in using the expression '*κύνα λυσσητῆρα*,' already referred to, the author of the Iliad much more probably referred to dogs infuriated with anger than to dogs suffering from Hydrophobia. All who are practically acquainted with the disease will admit that whilst on the one hand it is most improbable that the snappish ill-temper of rabid dogs should have been referred to, as a point of comparison, by Homer in the passages quoted by authors, there is on the other hand nothing more natural than that he should compare an angry man to a furious dog, inasmuch as there is nothing more suggestive of ungovernable passion than the frenzy of an infuriated (but not *rabid*) dog.

Whatever may have been the primary meaning of the word, there can be no doubt that, in the course of time, the word *λύσσα* and its very numerous derivatives—*λυσσάω*, *λυσσαίνω*, *λυσσηδόν*, *λυσσήεις*, *λυσσητήρ*, *λυσσώδης*, *λυσσόδηκτος*, *λυσσητικός*—were employed to express Hydrophobia, and in this sense the word *λύσσα* is employed by Plutarch and Dioscorides.

Hippocrates only alluded in the faintest manner, if at all, to Hydrophobia, and we agree with Dr. Bardsley in the opinion that, when speaking of the "*φρενέτικοι βραχυπόται*,"[5] the father of medicine probably only meant to treat of "a variety of phrenitis or mania."

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[1] See Thesaurus Grecæ Linguæ ab. H. Stephano construct. vol. iv. fasc. 2; also Damm. Nov. Lex. Græcum etymol. et real.; cui pro basi substratæ sunt concordantiæ et elucidationes Homericæ et Pindaricæ. This author derives *λύσσα* from *λύκος*, wolf, this animal being very often subject to Hydrophobia.

[2] Cyclop. of Pract. Med. vol. ii. art. Hydrophobia.

[3] Iliad, lib. viii. 299; ix. 237; xiii. 53.

[4] Plato, Herodotus, Sophocles.

[5] Prædict. lib. i. p. 69. Prænot. Lect. ii. p. 131.

Democritus, however, who was the contemporary of Hippocrates, if we are to believe the statements of Cælius Aurelianus,[1] was acquainted with the disease, and wrote upon its nature and treatment. "Etenim Democritus qui Hippocrati convixit, non solum hanc memoravit passionem, sed etiam ejus causam tradidit, cum de opisthotonicis scriberet." In two other passages in his interesting account of Hydrophobia, Cælius alludes to the opinion of Democritus as to the affection being one of the nervous system, and mentions the remedies which that author recommended for its treatment.

Aristotle was undoubtedly acquainted with the Hydrophobia of dogs. In his "Historia Animalium,"[2] besides describing it, he mentions that the disease is communicable to all animals but man. The opinion, although erroneous, points to the fact of his having directed attention not only to the characters but also to the mode of propagation of the disease. Both Asclepiades and Themison, according to Cælius Aurelianus and Dioscorides, were acquainted with Hydrophobia, and entertained peculiar views as to its nature. Themison is indeed said to have been himself subject to an attack of Hydrophobia, but as he recovered from the affection we may with probability conjecture that its nature was very different from that of true Hydrophobia.

Of the ancient writers, Celsus and Dioscorides, Cælius Aurelianus, Galen, and Avicenna, are those to whom we are chiefly indebted for our knowledge of Hydrophobia, and to their opinions we shall, on more than one occasion, revert. Since the period when they wrote, all systematic writers on medicine have treated of Hydrophobia. Foremost amongst those who have contributed to our knowledge on the subject are Van Swieten, Sauvages, Cullen, Fothergill, Mead, Trolliets, and Bardsley.

The recent history of Hydrophobia refers principally to Europe, not only because greater attention has been paid to this as to every other disease of men and animals in the Old World, but because the malady has been to a very great extent confined to Europe. We shall afterwards attempt an explanation of this fact, but in the meantime we may state that the oldest report of scientific interest that we can trace, refers to wolves afflicted with rabies, in Franconia, in 1271. Contrary to their usual habits, these animals spared the herds and flocks, and attacked shepherds in the fields, or followed human beings into the towns and villages. Upwards of thirty men fell victims to these attacks.[3] Canine madness prevailed considerably in Spain in 1500.[4] In 1590, we are told by Bauhin,[5] rabies raged in an epizootic form amongst the wolves in the province of Monthelliard. In 1604 it prevailed in Paris, and although it was doubtless common there, and after in various parts of Europe, we do not hear of it again until 1691,[1] when the heat of summer proved insufferable, the crops withered for want of rain, animals died in great numbers in Italy, and dogs went mad. This reference to the dependence of rabies on the hot season must simply be regarded as pointing to a coincidence. Severe outbreaks of rabies occurred in France and Germany from 1719 to 1721, in Hungary in 1722 and 1723, and atmospheric influences were blamed for similar attacks in various parts of the continent, from 1725 to 1726.[2] We learn, however, that wolves were affected as well as dogs at this period, and special mention is made of wild animals being seized with the disease in Silesia and Lusatia (now Bautzen, in Saxony). The malady was rife in London in 1760, in Philadelphia in 1779, in the West Indian Islands in 1783,[3] and in various parts of Europe from 1785 to 1789.[4] At this period special notice is again taken of rabid wolves which communicated the disease and terrified the people far more than even rabid dogs.

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[1] Op. cit. lib. iii. cap. xv.

[2] Hist. Animalium, lib. viii. c. 22.

[3] Addit. ad Lambert, Schafnaburg.

[4] Blaine's Canine Pathology.

[5] J. Bauhin, Memorab. Historia Luporum, 1591.

It was not till the commencement of the present century that foxes were discovered to suffer much and frequently from canine madness. In 1803 these animals were seen rabid in large numbers in the Pays de Vaud, in various parts of the Aubonne, Cossenay, Orbe, and Yserden districts at the foot of the Jura, and in 1804 similar outbreaks were witnessed on the northern shore of the Lake of Constance, and thence extending throughout the whole of Germany. Blaine tells us that in 1806 rabies in the dog became so prevalent in the vicinity of London that a day seldom passed without his being consulted on one or more cases of it, and sometimes he would see three, four, or five a day for weeks together. The kingdom of Wirtemberg and Grand Duchy of Baden were sadly ravaged by it in 1808 and 1809. Foxes continued to be affected, and these usually shy animals faced or followed men, cattle, or dogs in open daylight, bit them, and communicated the disease. The Wirtemberg Government ordered a report to be published on this subject in 1829, which contains particulars of these outbreaks.

From 1803 to 1820 is a memorable period in the history of Hydrophobia. In 1810 it spread rapidly through Southern Russia, and the same year it appeared in America, in the State of Ohio, where it destroyed dogs, wolves, and foxes, besides other domestic animals, and no small number of human beings. In 1815 the malady was raging in Denmark. Blaine tells us that in 1820 it was again on the increase in England, and for three or four years "continued alarmingly common," but moderated again for a few seasons. During the years 1819 and 1829, rabies was rife in Italy, and Brera speaks of a wolf which bit thirteen persons, of whom nine died hydrophobic. Wirth specially notices the period from 1819 to 1826 as remarkable for rabies amongst the foxes of Switzerland and Germany,[1] and the foxes communicated the malady to dogs, cats, horned cattle, horses, pigs, goats, and sheep. In 1824 rabies prevailed extensively amongst foxes, wolves, cats, and reindeer in Sweden, Norway, and even Russia. In the Rhine Provinces, various cantons of Switzerland, and in the kingdom of Wirtemberg, many men were bitten by mad foxes in the year 1827, but owing to the prompt measures usually adopted the individuals escaped; dogs and cats thus bitten, however, became rabid.

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[1] Ramazzini, pp. 157–186, and Baglivi (1828), vol. ii. p. 331.

[2] Breslau, Sammlungen, xxxiii. p. 90.

[3] Moseley's Treatise on Tropical Diseases.

[4] Schnurrer, ii. p. 389; and Fehr, Ueber die Hundswuth, Münster, 1739.

In 1830 the subject of rabies attracted great attention in England, and evidence on the subject was laid before a select committee of the House of Commons. Sir Benjamin C. Brodie, Professor Coleman, Messrs. William Youatt, Morgan of Guy's Hospital, Earle of St. Bartholomew's, Benjamin Travers, and others were examined. Some idea of the state of popular terror, if not of the number of bites by rabid dogs, may be gleaned from Mr. Youatt's statement in evidence, to the effect that he had applied lunar caustic to about 400 bitten people, and a surgeon at St. George's declared that within a limited time he had similarly operated on 4,000 without an accident. Mr. Youatt particularly referred to a great increase in the prevalence of the disease as contrasted with eighteen months previously. Mr. Henry Earle, of St. Bartholomew's, furnished unmistakeable evidence of the increased frequency of the disease in England, and stated that he had seen twenty-five cases of Hydrophobia in man in twenty-five years, whereas his father had only seen one in fifty years, before his time. It is certain from the statements of Blaine, Coleman, and others, that prior to 1830 foxhounds were affected in this country to an extent never witnessed before nor since.

That there has been a decided abatement in the frequency of the malady in England during the second quarter of the present century, as compared with the first, cannot be doubted, and it is believed that the law abolishing the use of dogs as beasts of burthen has operated greatly in favour of the change. That law not only led to a diminution in the number of dogs owned by a class of persons who could not keep these animals in a way calculated to prevent the spread of contagious diseases amongst them, but indirectly it led to the destruction of a lot of roving curs, kept by the poorer classes. The dog tax, too, which might have been beneficially enforced to a greater extent than it had been, no doubt tended to limit the number of valueless animals kept. Whatever may be the cause, it is certain that there is far less canine rabies, hence much less human Hydrophobia, in the British isles than on the Continent, and during recent severe outbreaks of this disease abroad little has been heard or seen of it amongst us.

Some remarkable statements have been published regarding Hydrophobia in France and the German States during the thirty-five years of comparative immunity which we have enjoyed. In 1830, when attention was attracted by the prevalence of the malady in London, there was occasion for alarm in Vienna, where thirty-nine cases of rabies in the dog were reported in rapid succession. Few instances of the disease were seen there for the succeeding seven years, but in 1838, 117 cases occurred; in 1839, sixty-three; in 1840, 317; and in 1841, no less than 141. Of the last number only thirty-one animals were affected with the dumb rabies. Only fifteen of the 141 were bitches. Two cases occurred in animals under one year, and only one in dogs between seven and fourteen years of age. The disease was specially rife in the months of February and May, in each of which twenty-one cases were observed; in January there were sixteen, seventeen in April, and eighteen in June. The fewest attacks were in the months of September, November, and December. From 1839 to 1842, rabies in the dog was very prevalent in Wirtemberg, and this was attributed by Professor Remy to communication from foxes. From the 1st of January, 1840, to the last of February, 1842, no less than 230 cases of rabies occurred in dogs, and twenty-one in bitches in the small Grand Duchy of Baden. In 1840-41-42, the malady was very destructive in Lyons and other parts of France. In the space of thirty years, no less than 779 dogs have died of rabies in the Lyons Veterinary School, giving a yearly average of nearly twenty-six cases. The largest number of these cases occurred in the months of June and of April. Only last year (1864), the city of Lyons was in great commotion owing to the terrible frequency of rabies canina.

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[1] Franque, Die Seuche unter den Füchsen und die ursprüngliche Wuth Krankheit der Hunde; Frankfort, 1827. Köchlin, Ueber die in unsern Zeiten herrschende Krankheit unter den Füchsen; Zürich, 1835. Wirth, Lehrbuch der Seuchen und ansteckenden Krankheiten der Hausthiere; Zürich, 1846.

Some idea may be formed of the great difference between Great Britain and the Continent in relation to the frequency of this disease, from the fact that having been more or less connected with veterinary colleges in this country since 1849, not a single case of rabies has come under our own observation here, whereas many instances have been seen by us abroad, on almost every occasion that we have visited foreign schools. When in Lyons and Paris in 1854, many were the cases we saw, and often did we see ladies unconsciously carrying their rabid pets to be visited by the professors.

Causes.—A glance at the history of Hydrophobia suffices to prove that, so far as man is concerned, the specific disease is due to only one cause, contagion. The poison is a fixed one, and flows from the sick animal's body with the abundant saliva which is secreted. It must penetrate the system to take effect, and is usually deposited by the teeth of rabid animals in and beneath the skin. It is not volatile, and the disease is therefore not infectious. Many circumstances affect its transmission, and on this we shall say more, after having spoken of the malady as it attacks the lower animals.

It is generally supposed that rabies originates spontaneously in the dog and other carnivorous animals. So deeply-rooted is this belief, that even men of eminence have attempted various explanations of the supposed fact. One of the oldest views refers canine rabies to the heat of the "dog-days" and the tortures of thirst. Another, recently revived, is that the malady is developed in dogs kept under restraint, and from indulging at proper seasons in sexual intercourse. These and other theories are so absurd, and so opposed to many of the facts which we must incidentally allude to in these pages, that we may state dogmatically they have no foundation in truth. Without entering into a discussion on matters of theory, we consider it more profitable to refer to the peculiar features of rabies in those of the lower animals which undoubtedly are capable of inoculating human beings with the rabid virus. We shall, therefore, speak of the disease as it occurs in the dog, wolf, fox, badger, marten, cat, horse, or sheep, pig, and goat.

Dog.—One of the facts of primary importance, in the history of canine rabies, is that it is far more prevalent in dogs than bitches. Professor Coleman stated in evidence in 1830, that on the occasion of rabies entering fox-kennels, the mad dogs bit dogs, but spared the bitches. We well know that, as a rule, rabid animals retain a certain affection for animals and people they know and care for. A dog will not bite its master at first, but rather avoids the presence of all he likes. There is some method in their madness, and we have no better proof of this than in the way in which bitches are spared by the rabid dog. Leblanc has published statistics, confirming the extraordinary prevalence of the disease in the males of the canine species, but he tried to bend the facts to demonstrate his theory that the disease is connected in its development with the repression of the desire for sexual intercourse. There are no experiments, no reliable facts indicating any ground for the belief that canine rabies originates in anything else than contagion, even in the dog; and the best British authorities, such as Blaine and Youatt, long since advocated this, the undoubtedly correct view of the subject.

It is singular that, even in relation to so active and certain a virus as that of rabies, we find instances of remarkable constitutional resistance to its effects. Thus when I visited the Lyons Veterinary College, in 1853, a pointer dog was in the infirmary which had been bitten no less than seventeen times by dogs affected with rabies, with which he had been purposely confined by Professor Ray. Other cases of a like kind have been recorded, but they may be considered rare and exceptional.

The period of incubation of the disease in the dog has been set down as between three and seven weeks. Blaine has met with a case in which rabid symptoms succeeded a bite in a week, though Youatt never saw a case with less than seventeen intervening days. In 1862 M. Renault made a communication to the Academy of Sciences at Paris, and stated that during twenty-four years he had made numerous experiments with a view to ascertain the period of incubation of Hydrophobia in the dog. During that period, 131 dogs have, under conditions which he describes, been either bitten by mad dogs, or inoculated with the foam as immediately collected from the mad animals. Of this number, 63 having presented no signs of disease during the four subsequent months, were not further observed. Of the 68 others, the Hydrophobia was developed at various periods, as shown in the following table:—

In	1 dog	between the	5th	and	10th	days.
	4 dogs	„	10th	„	15th	,,
	6 „	„	15th	„	20th	,,
	5 „	„	20th	„	25th	,,
	9 „	„	25th	„	30th	,,
	10 „	„	30th	„	35th	,,
	2 „	„	35th	„	40th	,,
	8 „	„	40th	„	45th	,,
	7 „	„	45th	„	50th	,,
	2 „	„	50th	„	55th	,,
	2 „	„	55th	„	60th	„
	4 „	„	60th	„	65th	,,
	1 dog	„	65th	„	70th	,,
	2 dogs	„	70th	„	80th	,,

There are no premonitory signs of an attack of the disease in the dog. When the period of incubation is passed, the animal is restless, dull, watchful, and snaps at dogs, other animals, or men, which come in its way. It shuns the light, but with much slyness seeks an opportunity of escape, and roves about town or country, manifesting extraordinary powers of exertion, and marked insensibility to blows and ill-usage. The habits of an animal may not change completely at first, and the recognition of persons it has been daily in contact with is sometimes very remarkable. The dilated pupils, the manner in which the eyes follow any object moved before them, and the peculiar modification in the bark, which is more of the nature of a howl, are amongst the most characteristic symptoms. The appetite is lost, thirst often considerable, and the animals usually drink without difficulty. The coat is staring, skin tight on the ribs, abdomen tucked up, head depressed, and nose protruded, with a dirty mouth and tongue, and sometimes a discharge of mucus and saliva from the sides of the mouth. In a certain number of cases, the nervous symptoms are very prominent at this period, and the lower jaw drops from paralysis of the muscles connected with it. The howl is then lost, hence the name dumb rabies for the cases associated with this symptom. Emaciation, craving after filth, which is swallowed with some difficulty, constipation, and a scanty discharge of high-coloured urine, are amongst the noticeable symptoms. Mad dogs have been known to eat portions of dead dogs, and mad bitches to devour their young. There is a singular absence of any marked acceleration of the pulse and breathing during the disease; the animal sinks, often paralysed in the hind quarters, and dies somewhat tranquilly from the fourth to the eighth day.

Schrader says that, of 267 cases of rabies, 223 belonged to the barking or raging variety, and 44 to the dumb or paralytic form.

Foxes show signs of the disease, as stated above, by losing all their shyness, following animals and men, biting them, losing their consciousness, and becoming paralytic, and otherwise presenting most of the characters of the disease in the dog.

Wolves are more to be feared than foxes, from their greater strength and ferocity. They attack human beings without fear, and usually bite them about the face, neck, and hands. As they approach death, they skulk away, and die in retired spots, blind, powerless, and in the quiet stupor seen in typhus.

Cats have scarcely less propensity to attack the uncovered parts of the bodies of individuals, and especially the face, than foxes. They scratch, whine in a hoarse manner, and die from the third to the fourth day.

Martens and *badgers* present most of the symptoms seen in cats and foxes.

In *the pig* there is a peculiar restlessness, squeeling grunt, disposition to bite, gaping, salivation, marasmus, paralysis of the hind quarters, and death from the fourth to the fifth day.

Rabies canina has been often observed in herbivorous quadrupeds, horses, oxen, sheep, deer, and other animals. The symptoms are marked and unmistakeable, presenting all the characters of the signs of rabies in the dog. From the imperfect powers of biting, communication of the disease from vegetable feeders to man is rare, and we need not, therefore, enter into further particulars on the subject here.

HYDROPHOBIA IN MAN.—As actual contact of the saliva of a rabid animal with a wounded or abraded surface is required for the development of Hydrophobia, we should expect that, of the total number of persons bitten, only a certain population should fall victims to the disease, and this is really found to be the case. Whether some individuals are, from idiosyncrasy, less prone to contract Hydrophobia than others, is not known, and it does not appear that age, sex, or constitution materially modifies the transmission of the disease. The situation and character of the wound affect, in all probability, the result, for it would appear that where the injuries are situated on the uncovered portions of the body, as on the hands or face, there is greater danger of the supervention of Hydrophobia than when they have been inflicted on the covered portions of the body or limbs. The clothing, in the latter case, protects the wound from the action of the saliva. The bites of wolves appear to have been more frequently followed by Hydrophobia than those of dogs; probably owing to the fact that, from their natural ferocity, the former animals, when attacking men, very commonly inflict severe injuries on the face and neck. Such circumstances as prevent the adoption of an energetic prophylactic treatment of those bitten by rabid animals, obviously favour the development of the malady.

The actual ratio of cases of Hydrophobia to the total number of persons bitten by rabid animals, has been very variously estimated by different writers on this subject. Whilst some have stated that only 5 per cent. of all persons bitten by rabid dogs become affected with Hydrophobia, others have estimated that 55 per cent. of the bites inflicted by rabid dogs are effectual in transmitting the disease. The ratio varies very greatly; probably between the limits we have mentioned. In the case of the bites inflicted by rabid wolves, the ratio of cases of Hydrophobia would appear to be as high as 60 per cent.

Symptoms of Hydrophobia in Man.—As it affects man, Hydrophobia presents symptoms which differ from those which have been described as characteristic of the affection in the dog. The chief point of difference is the almost constant occurrence in man of the peculiar spasms of the pharynx, excited when the patient makes an effort to drink, and which give rise to the dread of fluids, to which the disease owes its name. The cerebral symptoms appear to be less constant in man than in the dog; for—whereas amongst the most prominent of the symptoms in the dog is the thorough change in the natural instincts of the animal, and an uncontrollable tendency to attack and bite all around him—it would appear that in man delirium, although a frequent, is by no means a constant or necessary accompaniment of the affection.

Period of Incubation.—We are probably acquainted with no disease which possesses so long a period of incubation as Hydrophobia, or in which the period of incubation varies to so great an extent. This fact was well known to the earlier writers on Hydrophobia, one of whom most accurately remarks that whereas the affection usually supervenes within forty days after the infliction of a bite by a rabid animal, some are only affected after a year or more has elapsed.[1]

The disease has been very rarely developed sooner than eight days after the bite of a rabid animal, and rarely after a longer period than two or three months. The large majority of cases has occurred within four and eight weeks. Many authentic cases have occurred in which the period of incubation extended to eight or nine months, and a few where it appeared to be much longer. A remarkable case has been placed on record, in which a man who had been in prison for a period of more than two years, became affected with Hydrophobia, although he had only been bitten by a mad dog seven years previously.

During the period of incubation there is nothing which specially distinguishes the bite of a rabid, from one inflicted by a healthy animal. The wound may heal rapidly, or it may continue in an irritable condition, no peculiar train of symptoms occurring which can enable us to form a prognosis as to the probable issue of the case. The period of latency, or *delitescence*, as it has been termed, having elapsed, the following train of symptoms usually occurs. The wound becomes intensely painful, the pain often shooting from the extremities (if it be situated upon them), towards the trunk. If the wound have cicatrized, the cicatrix becomes red and irritable, or if, as is generally the case when the injuries inflicted by the rabid animal have been severe, it has not healed, it assumes an unhealthy appearance. With this local irritation, there is sometimes, though very rarely, some affection of the lymphatics of the part. In addition to the local phenomena, others of a constitutional character soon set in. The patient becomes peevish, irritable, and depressed; the skin is hot, the pulse rapid, and the appetite is lost. The peculiar depression of spirits just alluded to has been thought to be very characteristic of this, the first stage of Hydrophobia, and is indeed rarely absent. The unfortunate patient, who has been dreading the consequences of an accident of which the gravity is well known to him, often deludes himself with the hope, from the absence of all symptoms for a time, that all fear of danger has passed, and only awakens to a feeling of the real horror of his position when the local phenomena, which have been described, supervene. It is not strange, then, that they should be accompanied by a feeling of melancholy and impending evil.

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[1] Cæl. Aurel. lib. iii. cap. ix.

The first stage of Hydrophobia has been termed that of "recrudescence." Its characters are not fixed and invariable, for it has been noticed, in some cases, that no local symptoms occurred before the development of the true hydrophobic phenomena, and in others that melancholy and irritability were absent. In these cases, a feeling of general "malaise" and pyrectic phenomena have alone been present. This stage of recrudescence, of irritation and melancholy, as it has been variously styled, is usually of very short duration. After a few hours, or at most a day or two, the patient begins to complain of stiffness about the head and neck; and then the most characteristic manifestation of the disease sets in—an affection of the pharyngeal muscles, which throws them into the most painful spasms when the unfortunate sufferer attempts to eat, but specially to drink. The patient suffers from the most agonizing thirst, whilst the knowledge of the spasms which will accompany any attempts at drinking cause him to dread even the sight of liquids. "Miserrimum genus morbi; in quo simul æger et siti et aquæ metu cruciatur."[1] The hot and parched mouth becomes full of a clammy and viscid saliva which the poor sufferer is continually attempting to spit out, giving rise to the sound which the older writers compared to the barking of a dog. The heat of skin, and the frequency of the pulse, become excessive; the countenance is intensely anxious; the eye is bright and restless, and shuns the sight of a bright or luminous object, which often causes a fit of the painful spasms which, at first, attempts at deglutition only produced, but which afterwards the mere sight or even thought of fluids brings on. Delirium may be, and often is, absent; when present, it is often violent, and almost maniacal. In some cases a fit of general convulsions supervenes; in others, the spasms become weaker, and the patient, sinking into a state of comparative quiet, may regain, after a period of agitation and delirium, composure and quiet. Death may supervene early; it is often preceded by a stage of profound coma. A certain paralysis of the lower jaw, which drops, and allows the saliva to flow from the angles of the mouth, often marks the later stages of some cases of this sad disease, from which probably no one ever recovered.

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[1] Celsi Med. Lib. v. cap. xxvii.

The hydrophobic stage, which has just been described, is of short duration, death usually occurring before the end of the second day, life very rarely having been prolonged beyond the fourth day.

DIAGNOSIS.—In its first stage, when the phenomena of *recrudescence*, as they have been termed, are first manifesting themselves, it is impossible to form an accurate diagnosis. The feverish symptoms, and the fresh accession of pain in the bitten part, are symptoms which may, and often do occur in persons who have been bitten by animals not in a rabid condition—bites being very liable to heal slowly, to become the seat of neuralgic pains, and to give rise to considerable constitutional disturbance.

When Hydrophobia is fully developed, a careful study of *all* the phenomena cannot fail to lead to a correct diagnosis. It has apparently been confounded with diseases whose only resemblance to it consisted in the occasional occurrence of pharyngeal spasms, as inflammation of the larynx, trachea, and œsophagus. Hydrophobia may, however, more readily be confounded with hysteria, acute mania, and tetanus. A perusal of many of the recorded cases, especially of those which are said to have terminated in recovery, will readily convince the inquirer, that acute mania has sometimes been mistaken for Hydrophobia. In certain cases of this disease, it would appear that a dread of fluids has been manifested, whilst the general appearance of the patient has singularly resembled that of patients affected with Hydrophobia. The absence of the history of a bite, the history of premonitory symptoms of mania having existed, the duration of the disease, and the rapidity of the pulse—which in acute mania is very rarely above 90, whilst in Hydrophobia it is usually much higher—are the data upon which a differential diagnosis should be based.

French writers have described a definite disease under the terms "hydrophobie non rabique," "hydrophobie rabiforme," "rage spontanée," which is said to resemble in the closest manner true Hydrophobia. This affection, it is alleged, has generally been caused by fear, which has supervened after the bite of a rabid dog, or after communication with persons suffering from the disease. Its symptoms are, it is said, occasionally quite undistinguishable from those of true Hydrophobia; there is in the spurious form, however, very commonly a desire to bite. The patient may die in a few hours or days, or may remain affected for weeks or years. The diagnosis, it is stated, rests chiefly on the non-discovery of a part which has been bitten. We cannot see the propriety of admitting the existence of a non-rabid Hydrophobia, for the cases which have been described as examples of it may be more accurately classified as cases of either hysteria, acute mania, or true Hydrophobia, which had not been recognised.

Tetanus presents certain points of resemblance to Hydrophobia, tetanic spasms sometimes affecting the pharyngeal as well as the other muscles. The chief points of difference are, that Hydrophobia supervenes on a bite, whilst tetanus usually follows some other injuries; that the latter disease supervenes sooner after the injury than does Hydrophobia; that in tetanus the spasms are usually not intermitting; that trismus usually exists; that it is not marked by the distressing thirst, or the abundant discharge of saliva, which is so common in Hydrophobia; and that delirium very rarely occurs during its course.[1]

MORBID ANATOMY.—There are no cadaveric lesions which can be said in any way to characterise Hydrophobia. The most constantly observed phenomena have been the following:—

Redness and swelling of the fauces and gullet, occasionally accompanied by enlargement of the salivary glands, and sometimes with redness of the stomach. The trachea and bronchi have often been found injected, and filled with frothy mucus. The lungs are congested, occasionally inflamed, sometimes empty and œdematous. (Trolliet.)

The brain has been found occasionally congested; sometimes there has been effusion into the arachnoid and lateral ventricles. The medulla oblongata, and the origins of the seventh, eighth, and ninth nerves have been noticed to be congested, thickened, and softened.

PROGNOSIS.—When the disease is fully established, the prognosis is necessarily of the worst description, death being inevitable. Under this section a question of greater practical importance may be discussed, and the solution of which depends upon the facts which have already been referred to. A patient having been bitten by a rabid dog, when can the physician give a tolerably favourable opinion as to the issue of the case? when can he express a pretty confident opinion as to the safety of his patient? As the large majority of all those who are bitten by mad dogs escape Hydrophobia, even when no treatment is adopted, a favourable and encouraging, though a guarded, opinion can and should be given, even immediately after the accident. As the greater number of cases occur between the thirtieth and fortieth days, when the latter period is safely passed the prognosis becomes more and more favourable. After the end of the second month the large majority of patients may be considered safe.

THERAPEUTICS.—Since Hydrophobia was first described, all authors have devoted considerable attention to the treatment of the disease, both prophylactic and curative. It has long been known that the evil effects of the bite of the mad dog are probably often prevented by the adoption of an active local treatment of the bitten part. Celsus accordingly recommended the application of the cupping glass to the bitten part, or even the employment of the actual cautery. Since his time, in addition to the supposed specific methods of prophylactic treatment, nearly all authors have recognised the use of local treatment. Excision of the bitten part, the application of the actual cautery, or a combination of excision and cauterization; the application of various caustics, amputation of a bitten limb above the seat of the injury, have all been suggested and employed by different surgeons, and there appears to be good reason for believing that good effects have followed all these various methods.

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[1] Holmes' Surgery, vol. i. p. 313, art. "Tetanus," by Mr. Poland.

Complete and early excision of a bitten surface, as soon as possible after the infliction of the injury, is the method which has found most general favour in this country, and is perhaps the best which can be adopted. When, from the situation of the wound, excision is inadmissible, recourse should be had to the free use of caustics, and of these we should prefer the most powerful at our disposal—such as, presumably, would decompose such an animal virus as that of Hydrophobia; potassa fusa, or strong nitric acid, should have the preference. It must be mentioned, however, that, in the opinion of some, recourse need not be had to such severe methods. The late Mr. Youatt, who in the course of a long experience had treated a very large number of persons who had been bitten by dogs undoubtedly rabid, placed the greatest reliance upon cauterization with solid nitrate of silver, which in his experience had in every case prevented the development of Hydrophobia.

Besides the local modes of treatment, the older physicians believed in the efficiency of other remedies. Prolonged and sudden immersion in cold water was from the remotest times considered of great value in warding off a fit of Hydrophobia, and was practised to within a comparatively recent period. Celsus speaks of the plan with much favour, and Desault, in his well-known treatise on rabies, describes the mode in which the operation was performed in his days. It is in the memory of persons still living, that those bitten by mad dogs were, in England, taken to the sea-coast and dipped in the sea.

From the fact that so few of those bitten by rabid animals actually contract Hydrophobia, we should expect that a large number of specifics should have been proposed for its treatment. A credulous physician who happened to have administered some remedy to a few persons bitten by a mad dog, finding that no evil consequences followed, and forgetful that had nothing been administered his patients would, in all probability, have enjoyed equally immunity, was only too ready to believe that he had at last discovered a specific for a terrible disease. Thus doubtless it was that the Ormskirk medicine, a compound of chalk, alum, Armenian bole, alecampane-root, and oil of anise; the Tonquin medicine, a mixture of musk and cinnabar; and the Tanjore pills, a combination of arsenic and mercury, acquired for a time a wide and fictitious reputation.

The use of the hot-air and vapour bath has lately been recommended as a prophylactic in Hydrophobia, and it has been stated even to have cured the disease when fully developed. In the absence of any well-authenticated cases, we cannot decide upon the value of the remedy. That it might help to eliminate a virus which lurks so long in the system before producing its specific effects, is, however, not impossible.

Amongst the most important questions for discussion, in considering the treatment of Hydrophobia, is the following:—In the event of the peculiar phenomena of recrudescence manifesting themselves after a very long interval in a person who has been bitten by a rabid animal, should any local treatment be adopted? Should the bitten part or the cicatrix which has formed be excised or amputated? This question cannot at present be answered positively. As, however, cases have undoubtedly occurred in which such a local treatment of a bitten part has, even after the always-to-be-dreaded symptoms of recrudescence had set in, appeared to prevent the development of Hydrophobia, we are of opinion that the local treatment, severe though it may be, should be tried.

In the treatment of Hydrophobia, when fully developed, we must rely entirely upon general principles of medicine in attempting to relieve the sufferings of the patient. No specific method of treatment has been shown to have the slightest influence in checking or modifying this disease, from which, in all probability, no one ever recovered. We should try to soothe and comfort the unfortunate patient in every way in our power, to attempt to allay the great nervous excitement by means of sedatives, to support the failing strength by stimulants, and chiefly to prevent all noises, draughts, and other sources of excitement, which are so liable to bring on the painful pharyngeal spasms. Desault suggested, and apparently with good reason, that large fluid enemata might with advantage be administered. By checking the agonizing thirst they would, in all probability, greatly lessen the sufferings of the patient.

ENTERIC OR TYPHOID FEVER.

By John Harley, M.D. Lond., F.L.S.

Definitive Description.—A continued febrile condition of uncertain duration, accompanied by marked intestinal derangement, and invariably associated with lesion of the solitary and agminated glands of the intestines. It commences in anorexia, with nausea or vomiting; its progress is marked by profuse diarrhœa of light ochre-coloured watery stools, associated with abdominal pain, tenderness, and tympanitic swelling; and, if the issue be unfavourable, it terminates in exhaustion, intestinal hæmorrhage, or perforation of the bowel. Death usually occurs in the fourth week. In the early period, the disease is attended by more or less pyrexia; as soon as it is fully developed, there is well-marked hectic fever. During the height of the disease a scattered papular rash appears in successive crops on the abdomen and chest. The rapidity with which the symptoms are manifested, and the degree to which they are developed, vary greatly in different cases. The intestinal disease is frequently obscured by the concurrence of pulmonary or cerebral complications.

Synonyms.—*German*—Abdominal Typhus, Darm-typhus, Typhus gangliaris vel entericus, Ileo-typhus. *French*—Fièvre Typhoïde, Entérite septicémique, Fièvre muqueuse, Fièvre entéro-mesentérique, Gastro-entérite, Dothiénentérite, Entérite-folliculeuse. *English*—Typhoid Fever, Autumnal or Fall Fever, Slow Nervous Fever, Common Continued Fever, Hectic Fever, Infantile Hectic Fever, Infantile Remittent Fever, Entero-mesenteric Fever, Gastric Fever, Enteric Fever, Intestinal Fever, Pythogenic Fever, Cesspool Fever. *Common*—Bilious Fever, Gastro-bilious Fever, Muco-enteritis.

The above are the principal synonyms in use. Many others could be given; but as they may be either recognised as modifications of the foregoing, or have no restricted application to the particular disease under consideration, they need not be mentioned here. All may be found in Dr. Murchison's valuable work on the "Continued Fevers of Great Britain," p. 385, *et seq.*

The appellations "Typhoid," "Abdominal Typhus," and the like lead to an association of two diseases in the mind, which does not exist in reality; such terms therefore lead to confusion. "Gastric" has reference to an organ which, at most, only functionally sympathises with the principal lesion; the term "Pythogenic," introduced by Dr. Murchison, to imply the putrid source of the disease, is, on the one hand, too general, since it may be argued, with equal reason, that other acute diseases, besides the one under consideration, arise from this cause; and, on the other hand, it is not sufficiently comprehensive, since it would appear that Enteric Fever may arise from other causes than putrid or sewer emanations.

In adopting a term to distinguish the disease, we would select one which at once marks it out from all others, and points to a constant feature. Such a term is "Entero-mesenteric," employed by MM. Petit and Serres, in 1813. This appellation is a brief definition of the disease, and but for its inconvenient length we would employ it here. Acknowledging the direct sympathy which the mesenteric glands have with the intestinal lesion, we prefer, however, to use the shorter term, "Enteric Fever."

Preliminary Observations.—No disease presents, in the mode of the accession of its characteristic symptoms, in the gravity and sequence of these, and in its whole course and ending, so many variations, irregularities, complications, and accidents as Enteric Fever. A complete and consequent history of the disease will be best obtained by considering: 1st, The symptoms attending its development and progress, the condition of the secretions, and the modes of termination, the accidents, and sequences of the disease; 2dly, The morbid anatomy; 3dly, The pathology; 4thly, The associated pathology, meaning thereby a comparative view of the development of the particular intestinal lesion in other acute diseases—an investigation of great importance in the comprehension of the relation of acute diseases generally, but one of especial value in the elucidation of the nature of Enteric Fever; 5thly, The varieties; 6thly, The distribution; 7thly, The causes; and subsequently, there will remain to be considered the Diagnosis, Prognosis, Therapeutics, and Prophylactics of the disease.

Clinical History of the Disease.

Development and Progress.—In considering the clinical history of Enteric Fever, it will be convenient, with reference to the mode of access and development of the symptoms, to group the cases into three classes, viz. (1) Those in which the symptoms of gastro-intestinal irritation remain latent for days, or even weeks, after the patient has declined in health; (2) Those in which gastro-intestinal derangement is the chief feature of the disease from its outset to its termination; and (3) those which, in the suddenness of the invasion, the severity of the symptoms, and in the rapid course of the disease, closely resemble cases of narcotico-acrid poisoning.

Many of the cases of Enteric Fever belong to the *first class.* The disease indeed usually commences insidiously, and without premonitory indications of intestinal disorder. The decline of his health has been so slowly progressive and uniform, that the patient cannot state precisely when his illness commenced. For days or weeks past he has lost appetite, and felt weak, languid, and disinclined for bodily or mental occupation, complaining of a little headache, chilly sensations, chiefly referred to the spine, and of weariness and pains in the limbs. His increasing weakness sooner or later compels him to relinquish his ordinary occupations, and to apply for relief. We find the tongue moist, and tolerably clean; the skin cool, pallid, and free from rash; the pulse is rather small, and slightly accelerated; the mind is clear, and the expression natural; the bowels have responded to a purgative, but now they are regular, or perhaps again constipated; the abdomen is natural; the other functions of the body are regularly performed. The patient may remain in this condition for some time, but sooner or later the nature of the disease is manifested by the appearance of its characteristic symptoms. At first, there is increase of the early symptoms, anorexia is aggravated to nausea, and sometimes there is vomiting of green fluid; the skin becomes hot and dry; the pulse is increased in frequency; the tongue is furred, and usually presents red prominent papillæ at the margins and tip; there is great restlessness and increased headache; the bowels become loose, and the abdomen is a little full, painful, and tender—the right iliac fossa especially so, and pressure upon this part usually produces gurgling. A few round rose-coloured papules may now be observed upon the abdomen, chest, or back. They disappear on pressure, and closely resemble the papules of variola during the first few hours of their existence; but they are not quite so large, nor so hard. Their number varies much, and the quantity of rash bears no proportion to the severity of the disease. Usually we do not find more than three or four papules; occasionally the chest and abdomen is closely spotted with them. In one case we observed them profusely scattered over the thighs, legs, and feet. During the prevalence of the diarrhœa a few fresh spots appear every day, and after forty-eight hours the old ones begin to fade away. Diarrhœa, frequently associated with bilious vomiting, now prevails, and the abdomen becomes distended, and in many cases more or less tympanitic; the alvine dejections are watery, and of a light ochre colour, and putrid odour. At first they are acid, but they soon undergo change, and become ammoniacal and have an alkaline reaction.

With the supervention of diarrhœa, all the symptoms become greatly aggravated; the pulse ranges between 120 and 130; the skin is often pungently hot, and occasionally attains, towards night, a temperature of 107 or 108°. During this stage there is great irritability, and often considerable delirium, especially at night. In some cases there is no delirium and the mind remains clear to the last. Symptoms of active, pulmonary congestion,—accelerated breathing, pain in the chest, mucous râles, and expectoration streaked with blood,—are also liable to arise. The aspect of the patient is usually indicative of suffering, but the countenance is clear, and the eyes bright, as in scarlatina; the cheeks are suffused with a hectic flush. The urine is clear and copious; it is frequently retained.

The patient may continue in this condition for several days, the body meantime undergoing rapid emaciation. The tongue may continue moist, in which case it becomes pale, large, and flabby, and is liable to ulceration about its margins, and the formation of deep fissures with everted margins across the dorsum. In many cases the tongue becomes dry, red, contracted, and fissured at this period. The mucous membranes become dry and inflamed; the gums are liable to bleed, epistaxis frequently appears, and sordes begin to form upon the dry teeth. Here is the turning point of the disease. If we can subdue the gastric irritation, and keep food in the stomach, and restrain the diarrhœa, the symptoms will usually take a favourable turn. The abdominal pain and hectic fever diminish; more nourishment is taken; the tongue begins to moisten at the edge, and the cracks to heal; the rough cuticle, especially that of the abdomen, to desquamate. Sometimes sweating is re-established suddenly, and with the appearance of a copious eruption of sudamina over the chest and abdomen; the bowels may continue loose, but the stools are of a darker, greener colour. At this stage a relapse is very common—the diarrhœa, vomiting, and hectic returning with the former severity. The patient is not free from the danger of a relapse, until the stools have become solid. The improvement is slow, and, the diet being restricted, the emaciation persists for weeks. The desire for food is usually great; the digestive function is ultimately completely restored, and the patient regains his former weight and strength.

When the case tends to an unfavourable issue, the diarrhœa continues unchecked; the abdominal pain, and usually the tympanites also, increase; the exhausted patient lies motionless upon his side or back, drowsy or apathetic, and uttering feeble moans; the knees are drawn up, and his pinched, flushed countenance manifests pain on the slightest disturbance; the skin is pungently hot, the pulse very fast and thready, the teeth and tongue are blackened with sordes, the continuous delirium lapses into coma—the typhous condition is complete. Watery stools are passed involuntarily, the patient hourly sinks, and at last quietly expires. As soon as the diarrhœa appears, and as long as it continues, the patient is liable to intestinal hæmorrhage. The blood may appear repeatedly and in considerable quantity day after day in the stools, or the patient may become suddenly blanched and die of syncope, without any discharge of blood *per anum.* In such a case the intestines will be found distended with blood.

Hæmorrhage, however, is not the only accident we have to anticipate. The patient is often cut off by perforation of the bowel. This dreaded event may be expected if, with a persistence of the diarrhœa, the tenderness and tympanites increase, and vomiting and hiccup supervene. Perforation is most commonly preceded by symptoms of general peritonitis accompanied by excessive tympanites, persistent hiccup, and vomiting. A paroxysm of more intense abdominal pain sometimes indicates the occurrence of this fatal result.

As an illustration of the insidiously progressive class of cases, I give the following outline of the history of a patient who came successively under the care of Dr. Murchison, Dr. Buchanan, and myself, in the London Fever Hospital.

Case 1.—C. Bushell, aged 24, a well-nourished, dark-complexioned woman, experienced a feeling of lassitude, accompanied by chilliness, pains in the limbs, and slight headache, with loss of appetite, for about *four* days. She took to her bed on the *fifth* day, and was admitted on the *sixth.* A mild attack of typhus was suspected, and from day to day the tongue, pulse, surface of the chest and abdomen, and the nature of the secretions were examined. Still no positive disease declared itself, and no diagnosis was made. The pulse was 80–84, tongue natural, skin not hot and free from rash, bowels not acting every day, abdomen natural. During the time she remained in the hospital she exhibited no new symptom, complaining only of weakness, chilliness, general pains, and want of appetite. She ate fish, and subsequently meat, and was kept in bed during a portion only of the time of her sojourn in the hospital. As she was apparently suffering from mere debility, and had improved a little, she was discharged on the *fifteenth* day. She was again admitted on the *twenty-ninth* day, and stated that she had not been well since she left the hospital, having still suffered from excessive weariness and pains in the limbs, headache, and chilliness, followed by a little feverishness. Lately she has had shivering, the bowels have been rather constipated, she has lost all appetite, and feels rather sick; pulse 120, feeble; tongue moist and white; no rash; no cerebral or pulmonary symptoms. *Thirty-first* day: tongue clean and red at edges, a moist thick fur on centre; bowels became rather loose, and six or seven rose-coloured papules appeared on the abdomen; pain and gurgling in the right iliac fossa; pulse 120; skin hot; face flushed; sleeps badly. Day after day, to the *thirty-eighth* day, the purging increased, and fresh rose papules appeared; the abdominal pain increased; the tongue became dry, brown, and cracked; the pulse rose to 164. She died exhausted on the *thirty-ninth* day.

Autopsy.—Rotundity of the body preserved; lungs healthy, excepting engorgement of one lobe. Stomach, duodenum, and jejunum appeared healthy. The solitary and agminated glands of the lower part of the ileum swollen and inflamed; those near the valve were ulcerated and sloughy, and formed almost one continuous surface, raggedly disintegrated, and greatly swollen, extending around the whole of the last two inches of the bowel. A few of the solitary glands of the cæcum and ascending colon were inflamed and ulcerated. The corresponding mesenteric glands much swollen, congested, and softened. Spleen soft, twice its normal size; liver enlarged and fatty, weighing three pounds nine ounces (avoir.). Gall bladder distended with pale brown, watery bile, of excessively acid reaction and sulphuretted odour.

The *second class* of cases are perhaps the most frequent. In these the nature of the disease is manifest in the beginning. The patient may have felt a little indisposed previously; but he is in the midst of his usual occupations, or upon a journey, when he is overtaken with headache, shivering, and purging, followed by general pains and more or less pyrexia; there is complete anorexia, and nausea and vomiting are frequently amongst the earliest symptoms. There is pain in the abdomen, and great thirst. The prostration of the strength is very great, and the patient soon takes to his bed; the bowels continue to act two or three times a day, and the febrile symptoms and abdominal pain and tenderness persist; the tongue is moist, and usually coated with white fur; the edges and tip are red, and exhibit prominent fungiform papillæ. On the seventh day, or a little later, a few rose-coloured papules appear upon the abdomen, chest, or arms; the belly is a little full; there is great tenderness and gurgling in the right iliac fossa; the patient is unable to take food, and is distressed by occasional vomiting of bilious fluid. The fever runs high, there is great restlessness by day, and broken sleep and delirium at night. Great pains are complained of in various parts of the trunk, the hepatic and splenic regions are tender, and there is increased dulness in the latter, indicating enlargement of the spleen. The breathing is often quick; there is some cough; and evidence of the presence of active congestion, or of acute inflammation of the lungs, is rarely wanting at this or a little later period. The bladder is very liable to become distended at this stage. These symptoms may persist with greater or less severity for the next week or ten days, the patient passing from two to six watery ochre-coloured stools, containing a few shreddy flocculi, every day. The tongue becomes dry, with red irritable edges and tip, and elsewhere covered with a yellowish-brown cracked fur. If the patient have escaped the dangers of hæmorrhage and perforation, he may, at the end of this time, begin to progress towards recovery, or, if the symptoms take an unfavourable turn, he will almost surely die. When the purging has persisted for weeks, the days are critical. The following is a common case of Enteric Fever, beginning apparently in ordinary diarrhœa:—

Case 2.—E. R., aged 19, a well-nourished healthy woman, taken while on a journey of pleasure with shivering and purging, followed by headache and general pains. The bowels had been regular previously. The purging and other symptoms continued, and she became slightly feverish, and lost appetite. She took to bed on the *fifth* day of her indisposition, and was admitted into the hospital on the *ninth*, presenting the following symptoms:—Pulse 104, full; skin hot; tongue moist, and coated with white fur, which is disposed to form cracks; bowels very loose; motions fluid, light, ochre-coloured; abdomen tender, three distinct rounded and elevated rose-coloured papules here and there; there is great thirst, and the patient is very feverish and fretful. *Tenth* day: tongue very thickly coated, cracked in the centre, clean and red at the tip and edges; bowels very loose; stools of greenish fluid; twelve or thirteen fresh papular spots on abdomen. *Eleventh* day: pulse 96; tongue moist and superficially fissured; bowels still very loose; twenty-two fresh spots on abdomen; great pain across the abdomen and round the back. *Twelfth* day: tongue dry and brown at the tip and down the median line, aside of which it is covered with a thick crust of cracked yellowish-white fur; sides are moist and clean; stools frequent, copious, of light yellowish-brown fluid, possessing an acid reaction, and containing ragged yellowish flocculi; abdomen a little full; great tenderness and gurgling in the right iliac fossa; some fresh spots. *Sixteenth* day: the patient is emaciating very rapidly; pulse 90; tongue dry and red, devoid of fur except at base; purging a little diminished the last few days; yesterday's and this morning's stools together darkish-brown, fluid, alkaline; retention of urine, three pints drawn off; a few fresh spots, most of the old ones having faded and disappeared. *Eighteenth* day: no fresh spots; pulse 104, feeble; passes urine spontaneously. *Nineteenth* day: pulse 120; tongue dry, somewhat contracted, covered with a thin, dry, cracked, yellowish crust; much pain in the back and belly; cannot lie on the back "because it hurts her breath;" respirations 26; some fine crepitation at both bases behind; abdomen very tender; only two or three spots now visible; one copious ochre-coloured stool this morning. *Twentieth* day: pulse 136, feeble; one copious light-brown watery stool; skin cooler; sleeps well; has been sick two or three times. *Twenty-third* day: is much better; pulse 100; tongue clean and moist, excepting a dry median streak; bowels not opened for two days; the rash has wholly disappeared; hunger. *Twenty-fifth* day: pulse 80; bowels act once in two days; stools light, fawn-coloured, semi-solid; abdomen natural, bears moderate pressure; tongue moist, but furred; hunger; to take solid food for the first time—fish and bread. *Thirtieth* day: slight relapse to-day; pulse 108; anorexia; thirst; pains in limbs; headache; skin hot; abdomen painful; no action of the bowels to-day; no fresh rose spots. *Thirty-first* day: increase of the feverish symptoms; pulse 126; a little diarrhœa; stools light yellow; a copious eruption of sudamina upon the abdomen. From this date she continued to improve, and was convalescent on the *fortieth* day.

In the *third class* of cases the symptoms are so sudden and severe that there may be suspicion of poisoning by some acrid narcotic, such as colchicum or poisonous mushrooms. We find the patient in a state of high fever; there is intense heat of the head; acute delirium; frequent vomiting and purging; the tongue is red and dry; the abdomen tense and painful. We learn that his illness commenced a few days ago, with vomiting, purging, and great headache. The patient lapses into a state of stupor; the diarrhœa persists, and he soon passes into the typhous condition, and dies on the eighth or fifth day, or even earlier. The following is a good example of this class of cases:—

Case 3.—Alfred S., aged 20, a powerful well-developed man, was admitted into the London Fever Hospital, October 7, 1865, in a state of stupor, pulse 156, very feeble, tongue dry and brown, conjunctivæ injected, head hot. He lay prostrate, passing liquid stools involuntarily, and died comatose twelve hours after admission; there were no rose spots or other rash upon the skin. His friends stated that he was suddenly taken ill with sickness and purging, followed by fever and delirium. The matters voided were of a bilious character.

Autopsy.—Body well nourished, skin clear. *Head*—meninges, and brain, quite healthy; the ventricles and theca vertebralis contained only one ounce and a half of serum. *Chest*—lungs engorged, weigh three pounds, everywhere crepitant. Heart healthy, small clot in right ventricle. *Abdomen*—liver weighs three pounds three ounces, softish and flabby, a little fatty; gall-bladder distended with pale watery faintly acid bile of the colour of urine. Spleen enlarged, weighs fourteen ounces, natural in colour and consistence. Stomach slightly injected at the great end; duodenum and jejunum healthy; ileum of a violet colour externally; seven feet from the ileo-cæcal valve, a Peyer's gland, an inch long, was slightly swollen, and presented a prominent vascular elevation at one end. In the last six feet every Peyer's gland partially or wholly red, swollen, and reticulated. In the last four feet the glands were much elevated and the villous surface abraded; the larger patches were raised a fourth of an inch above the level of the mucous membrane; all were very soft, and exceedingly vascular, and of a fiery red colour. Between the Peyerian glands were innumerable solitary glands, forming elevations the size of a pea, surrounded by bright-red areolæ, and presenting yellowish unbroken apices. The intervening mucous membrane highly inflamed. The valve much swollen and deeply wrinkled. Cæcum and first foot of colon thickly strewn with swollen solitary glands as large as peas, having sloughy centres. A biliary calculus, the size of a kidney bean, lay at the lower end of the dilated appendix, which was healthy, excepting where one solitary gland formed a vascular elevation. A few of the solitary glands in the transverse colon were enlarged; with this exception, the large intestine was quite healthy. The follicular glands, at the root of the tongue, were injected and swollen. The mesenteric and mesocolic glands were enormously swollen, congested, and soft. The mucous membrane of the larynx, trachea, and bronchi was very red and covered with frothy mucus; the kidneys were congested; the bladder contained eight ounces of clear urine.

The two following cases illustrate the difference in the progress, termination, and effects of the disease in different individuals under the same general conditions:—

Two young men—J. Bennett and C. Beale—of the same age, and equally strong and well nourished, and resident together in a house in the immediate vicinity of King's College Hospital, were taken ill with febrile symptoms, the former on the 14th of August, 1865, the latter a week afterwards. Both patients died; Bennett on the 12th of September—the thirtieth day of the disease; Beale on the 13th of the same month—the twenty-third day of his illness. The rose spots were not developed in either case.

Case 4.—Bennett was admitted into King's College Hospital on the *fourteenth* day. He stated that he was attacked with headache and shivering, followed by sweating, general muscular pains, and sore throat. An aperient produced a loose state of the bowels for a day or two. Deglutition was very painful for three or four days. He got better, but remained very feeble, and did not recover his appetite. On admission he was pallid and weak, the throat had recovered, the tongue was moist, and the pulse but slightly accelerated; there was no diarrhœa, no rash, no abdominal pain or tenderness. He appeared to be suffering debility from a previous febrile attack. He continued to improve, regaining a little strength and appetite, and was discharged at the end of a week.

On leaving the hospital he went to his work, but soon felt too weak and ill to continue it, and after three days he again applied for advice, and was readmitted into King's College Hospital on the 8th of September, when he came under my care. He was dull, peevish, and prostrate; since he left the hospital the bowels had been loose. At this date there was moderate diarrhœa. The face and skin were pallid, the latter hot, perspiring, and free from rash; the abdomen slightly tympanitic and tender. Pulse 108. Tongue dry and brown, covered with a thick cracked crust. Respirations 42; slight dulness, and fine crepitation over the back of the chest. During the next four days he lapsed into stupor, and lay on his back with the eyes closed, the knees a little drawn up, moaning occasionally, and picking with his fingers, the wrists and forearms being constantly twitched. The diarrhœa was soon checked by sulphate of copper and opium, but he resisted when attempts were made to open the jaws and administer drinks; the pulse and respirations increased, and he died comatose, four days after admission, on the *thirtieth* day of the disease.

Necroscopy nine hours and a half after death.—Body somewhat emaciated, viscera warm, blood fluid. *Chest*—lungs congested, bronchi much injected, two yellow masses of solid matter, the size of peas, like tubercle, in the lower lobe of the right lung near the border. Heart healthy, contained a pale soft clot. *Abdomen*—liver enlarged, weighed three pounds nine ounces; bile, pale, watery, small in quantity. Spleen, of natural consistence and colour, weighed ten ounces and a half. Mucous membrane of the large end of stomach much congested. Peyer's glands of the upper portion of the ileum swollen, vascular, and reticulated; all those, and great numbers of the solitary glands in the lower four and a half feet of the ileum, greatly swollen and superficially ulcerated, the larger glands forming fungous elevations, with margins raised a fourth of an inch above the level of the thin wall of the bowel, and resembled large indurated chancres. Fig. 9 (p. 362) represents one of these glands situated at a distance of 15 inches from the ileo-cæcal valve. The centres were slightly depressed, and stained of a dirty greenish-brown colour. The swollen glands were firm and transversely wrinkled. The solitary glands formed smooth rounded elevations, the greater number corresponding in size to the tips of the fingers; each one presented a firmly adherent central slough. Cæcum healthy, but the solitary glands throughout the rest of the large intestine, including the upper part of the rectum, formed sloughy elevations like those of the ileum. In the sigmoid flexure there were fifty-four such elevations. In the transverse colon only six. In the ascending colon they were as thickly strewn as in the sigmoid flexure. The mesenteric and mesocolic glands were greatly enlarged, vascular, and softish. The brain was not examined; all the other organs were healthy.

Case 5.—Beale was admitted under my care into the London Fever Hospital on the 1st of September. His illness commenced a week previously with anorexia, cold chills, headache, sickness, pain in the bowels, and diarrhœa. *Eighth* day: pulse 96; tongue moist and furred at the margins; skin pallid and hot, no rash, no headache; mind quite clear. Abdomen slightly distended; gurgling in the right iliac fossa. *Eleventh* day: bowels became very loose, and the abdomen tympanitic and tender. *Twelfth* day: pain in the abdomen; in the evening bowels very loose. *Eighteenth* day: febrile condition continues; pulse 108 to 120; tongue moist and furred; skin hot, free from rash; face very pale. The abdominal symptoms—diarrhœa, tympanites, and abdominal pain—have daily increased in severity since the twelfth day, and to-day there is evidence of general peritonitis; six leeches were applied to the right iliac fossa. *Nineteenth* day: leeches caused profuse bleeding, which was stopped with difficulty by the application of nitrate of silver; pulse 132, weak; tongue dry and brown; bowels quiet. He gradually sank, and died on the *twenty-third* day of his illness, retaining a clear intellect to the last.

Autopsy.—Body somewhat emaciated. *Chest*—lungs weighed fourteen ounces, floated in water, contained a dirty-brown fluid. Heart healthy, contained a colourless clot in the right ventricle. *Abdomen* displayed the effects of general peritonitis, the lower part of the cavity contained about a quart of turbid serum, and the coils of the small intestine were adherent to each other, and to the lower part of the abdominal wall, by layers of solid lymph. Liver weighed three pounds and a quarter, soft, friable, and fatty. Bile moderate in quantity, of light ochre colour, watery, and very acid, instantly turning blue litmus paper bright-red. Spleen weighed ten ounces, of natural colour and consistence, but flabby. Intestines distended; on separating the purple adherent coils of the ileum, a perforation a fourth of an inch in diameter was discovered six inches from the cæcum; the opening in the intestinal wall was plugged with the solid lymph that adhered to the contiguous coils of the bowel, so there was no escape of fæcal matter into the peritoneal cavity. Stomach, duodenum, and jejunum healthy. Intestines contained some smooth, soft, formed fæces, varying in colour from light ochre to dirty white. Mucous membrane of the ileum uniformly red and inflamed, covered over with tenacious firmly adherent mucus, of a bright ochre colour. The solitary and agminated glands of the upper portion of the ileum quite healthy; lower down they were vascular and swollen; two feet from the cæcum the first signs of ulceration, and in this last portion of the ileum the solitary glands were swollen to the size of a pea, and presented ragged excavated centres. The last twelve inches contained several Peyer's glands in a ragged state of ulceration, the ulcers having raised, firm, very vascular, and angry-looking edges, and irregular depressed surfaces, formed apparently of yellow sloughs, adhering to a raw, almost bleeding surface, beneath. These sloughs could be readily separated with the finger-nail. Their lower surface had a yellowish colour; they were friable, and some parts had an almost cartilaginous consistence and paler colour. After washing and careful examination these sloughs were found to be composed of solid lymph, agreeing precisely in physical and microscopical characters with the solid lymph which adhered to the corresponding peritoneal surface of the bowel. The harder and whiter portions were composed of lymph contained in the meshes of the areolar tissue of the gland, and were, therefore, really sloughs. The more advanced ulcers were seated on the inflamed and thickened muscular layer. The perforation corresponded to the centre of one of the large ulcers. The cæcum, colon, and rectum, free from inflammation and perfectly healthy throughout, and the solitary glands inconspicuous. Mesenteric glands greatly congested and swollen; those lying in the angle of junction between the large and small intestine, the size of pigeons' eggs. Pancreas hardish, but apparently healthy. Bladder empty, healthy, as were the remaining viscera.

These two closely-associated cases are interesting, as illustrating the influence of constitution upon the progress of the disease. Bennett died in a typhous state from nervous complication, and with an amount of intestinal disease at least six times greater than that to which Beale succumbed a week earlier. Yet the intestinal disease in Bennett's case was latent to within five or six days of his death; the solid thickening of the affected glands (see Morbid Anatomy) forming, and promising to continue to do so, an effectual security against perforation. One of the parents of this young man died of consumption, and he himself had evidently been affected with syphilis.

Condition of the Alvine and Urinary Excretions in Enteric Fever.—(*a*) *The Stools* are remarkable for their fluidity and the absence of healthy bile; they have a pale ochre or drab colour, and a sickly, offensive odour. On standing, a flaky matter is deposited, composed of epithelium, disintegrated sloughs from the intestinal ulcers, and undigested particles of food. According to Dr. Parkes (Med. Times, June 1850, p. 396), the supernatant liquid has a specific gravity of 1,015, and contains about 40 parts in 1,000 of solid matter, consisting chiefly of albumen and soluble salts, particularly chloride of sodium. The stools are already in a state of decomposition, and after standing a short time are almost invariably alkaline. Immediately after they are passed they often have a neutral and sometimes an acid reaction. The offensive ammoniacal fluid contains much triple phosphate.

If salts of bismuth, lead, silver, or copper, have been administered, the dejections have a dark greenish-brown, or black colour.

(*b*) *The Urine* in Enteric Fever does not differ appreciably from that excreted in other inflammatory diseases. On the first accession of the febrile symptoms its quantity is usually diminished, but afterwards it becomes copious. As in all other febrile states, the chlorine is greatly diminished and the urea and uric acid increased. The chlorine is often reduced to a mere trace. The quantity of urea and uric acid excreted, appears to be proportionate to the degree of fever; when the pyrexia is at its height, the quantity of these constituents excreted in twenty-four hours is usually doubled, sometimes trebled. As the fever declines the quantity of urea and uric acid diminish to the normal quantity or below it, while the chlorine reappears more slowly. In case 2, sixty ounces of darkish-coloured, clear, acid urine were drawn from the bladder on the *sixteenth* day. After standing twenty-four hours it was quite bright and free from deposit; specific gravity 1024. One fluid ounce contained a quantity of chlorine equivalent to 0·36 grain of chloride of sodium, 14·8 grains of urea, and ·3 grain of uric acid: or, in the sixty ounces, 22 grains of chloride, 889 grains of urea, and 18·9 grains of uric acid. On the *twenty-first* day, when the febrile symptoms began to subside, the urine was copious and neutral; specific gravity 1016·4. A fluid ounce contained a quantity of chlorine equivalent to 3·9 grains of chloride of sodium, and 5·8 grains of urea. On the *twenty-third*, the urine was copious, of specific gravity 1010, clear, pale, and a fluid ounce contained a quantity of chlorine equivalent to 3·2 grains of chloride of sodium, and 5·8 grains of urea. A small quantity of albumen often appears during the height of the fever.

Occasional Symptoms and Accidents.—Peritonitis, local or general, is liable to arise whenever the ulceration of the coats of the bowel extends deeply towards the peritoneum. This membrane becomes highly inflamed in places corresponding to the bases of the ulcers, and from these circumscribed patches of inflammation the increased vascular action may spread and involve the peritoneum more generally, and produce considerable serous effusion. Perforation is occasionally prevented by the adhesion of the inflamed patch to a neighbouring coil or coils; and if it should occur after this adhesion has been effected, a circumscribed abscess, which may ultimately discharge itself into the bowel, is formed. Perforation frequently occurs, however, under less favourable circumstances, and the fæcal matter is extravasated into the peritoneal cavity. Sudden increase of pain, accompanied by vomiting, and soon followed by cold, clammy sweats, and collapse, announce the nature of the accident. Sometimes sudden collapse alone is the only indication of this fatal issue. In other cases the perforation has taken place so gradually, the aperture formed is so small, and the extravasation so inconsiderable, that the symptoms of peritonitis come on and attain their maximum very gradually, and without any sudden increase in the severity of the symptoms.

Perforation of the Bowel usually occurs within six inches of the ileo-cæcal valve, and in almost every case it is the small intestine which is perforated. Next to the lower end of the ileum, the cæcum is most liable to perforation. "Out of 435 autopsies recorded by Bretonneau, Chomel, Montault, Forget, Waters, Jenner, Bristowe, or made at the London Fever Hospital, perforation was observed in sixty cases, or 13·8 per cent." (Murchison, p. 511.)

Tympanites is present to some degree in almost every severe case. It usually comes on a week or nine days after the purging sets in. When excessive, it is a very grave symptom. It usually precedes perforation.

Intestinal Hæmorrhage is a frequent accident in severe cases. It was observed in twenty-nine out of 139 cases observed by Murchison, Louis, and Jenner. It is a grave symptom, inasmuch as it generally indicates deeply-extended ulceration. The hæmorrhage, however, frequently has its source in the congested capillaries of the common mucous surface, near the junction of the large and small intestines. The blood is never much changed. If the intestinal fluid be acid, it will be dark. The quantity of blood passed from the bowel does not always indicate the amount of the hæmorrhage. In the case of a young girl which I witnessed, under Dr. Todd's care, in King's College Hospital, a trifling hæmorrhage appeared, and shortly after, death occurred from syncope. The small intestines were found distended with red, clotted blood.

Retention of Urine is frequently present at the height of the early pyrexia. This condition cannot be overlooked for many hours unless there be considerable delirium.

Pregnancy.—Abortion is almost certain to occur if a pregnant woman be attacked with Enteric Fever. The only two pregnant women who have come under my care aborted, the one at the third month of gestation, the other at the fifth. Both recovered well. *Phlegmasia dolens* is apt to be a secondary complication in such cases.

Sequelæ.—*Marasmus* is the necessary attendant and consequent of extensive and prolonged disease of the mesenteric glands. After morbid action has ceased in these, they often become atrophied, and remain for a long time in a shrivelled, flaccid condition. In some cases the digestive and assimilating functions remain so defective that the patient fails to regain appetite and flesh, and slowly starves to death.

Imbecility.—Patients who continue long in a state of extreme emaciation commonly manifest proportionate defect of mental power. They become forgetful and apathetic.

Tubercle of the Lung is considered by some physicians to be a common sequel of Enteric Fever. Many cases presenting such an apparent sequence may be regarded as instances of tuberculosis *ab initio.* See "Associated Pathology" of Enteric Fever.

Partial Anasarca, unassociated with albuminuria, is an occasional sequel of Enteric Fever in enfeebled constitutions. *General Anasarca* is rare. A scrofulous girl, E. Gain, aged 18, lately came under my care in the London Fever Hospital, with well-developed Enteric Fever. General anasarca suddenly appeared on the thirty-fourth and thirty-fifth day of the disease, when the stools were solid, and she was convalescing favourably; she had not, however, left her bed. Œdema appeared simultaneously in the lower extremities, the face, and hands; it was preceded by acceleration of the pulse and increased heat and dryness of the skin, which was pallid, and rough from fine desquamation of the cuticle. The tongue was red and glazy, with very prominent fungiform papillæ—a condition which had existed throughout. The anasarca increased from day to day, and was associated with considerable ascites. The integuments of the abdomen and chest were very œdematous. At one time the eyelids were closed by swelling, and the patient altogether presented the same appearance as one labouring under an attack of acute dropsy after scarlatina. Simultaneously with the development of the anasarca, albumen appeared in the urine, and became very abundant. The secretion, however, retained its natural colour, and was normal in quantity, and, as long as the patient remained under my care, was free from renal casts or other deposit.

MORBID ANATOMY.

Wherever the source of morbid action in Enteric Fever may lie, its effects are constantly manifested in the small intestine, and it is upon the solitary and agminated glands of the lower third of the ileum that the disease usually expends its virulence.

Without positive evidence of inflammatory action in these glands, the disease would not be Enteric Fever. How far the converse of this—that inflammatory lesion of Peyer's patches is always due to a specific Enteric Fever—is true, will appear upon consideration of the Associated Pathology of the disease.

Morbid changes, consequent upon Enteric Fever, are found, (*a*) in the solitary and agminated glands of the intestine; (*b*) in the mesenteric glands; (*c*) in the spleen; (*d*) in the liver.

(*a*) *The Solitary and Agminated Glands.*—A Peyer's gland or "patch" presents in a state of health a variable number of rounded, shallow, concave depressions, averaging $\frac{1}{10}$th of an inch in diameter, and separated by narrow linear ridges of mucous membrane, running in from the general mucous surface and on a level with it, and forming a network, in the meshes of which—*i.e.* in the depressions—the so-called "closed follicles" lie. In death, after the ninth day, from Enteric Fever, we shall rarely fail to find these and the solitary follicles in every stage of inflammation.

At the distance of four feet from the ileo-cæcal valve we shall generally find Peyer's glands in their normal condition. Six inches nearer the valve we may find one in the earliest stage of inflammation; it is slightly swollen, and raised above the general level of the surrounding mucous membrane, and it is a little more vascular than in health.[1] On careful examination the swelling is found to implicate the network of mucous membrane chiefly; the ridges between the closed follicles are more vascular, wider, and more prominent than in health; and the intervening depressions are thus contracted and deepened, and the patch is more distinctly reticulated. The follicles themselves appear to remain unaltered; minutely examined under water, they have a dark, semi-transparent, violet-grey appearance, while the intervening ridges are injected with minute divergent blood-vessels. Seen at a distance, the patch is clearly distinguishable from the common mucous surface. The general appearance is that of a fine, pink or white, swollen network, with dark rounded meshes. Passing downwards towards the ileo-cæcal valve, each succeeding gland presents the above-described characters in a more marked degree, and the patches consequently become very prominent and distinct. Fig. 1 represents an agminated gland in this early stage of inflammation. It was situated thirty inches from the ileo-cæcal valve. The ridges were wide, prominent, and very vascular, and the depressions contracted and deep; at *a*, the swelling and contraction was greatest. Fig. 2 represents the next patch, nearer the cæcum. This gland was much swollen and soft, and formed a prominent, fungous-like projection of the mucous membrane. Its borders rose abruptly from the general mucous surface, and were smoothly rounded, devoid of reticulations, and slightly more elevated than the central parts of the patch. The ridges were greatly swollen, so as to convert the depressions into minute deep pits. The next stage consists in the breaking down of the swollen mucous membrane around the dark pits, and the formation of circular aphthous-like ulcers, each having for a centre a depression corresponding to a closed follicle. If this disintegration be general, the swollen gland soon presents a ragged, spongy appearance; examined under water, we find the irregular surface to be composed of a fine stroma of dirty, shreddy, fibrous tissue, containing a number of circular, rounded excavations: these are the follicles; they have not undergone further enlargement than slight thickening of their walls, which are thus rendered very distinct.

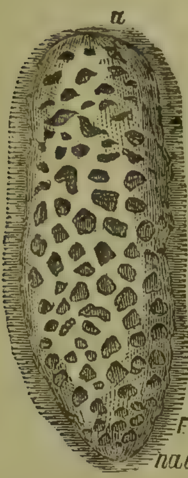

FIG. 1
nat. size

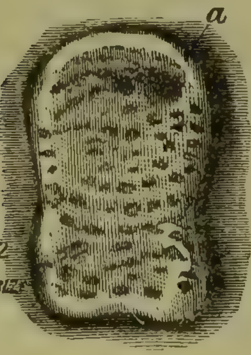

FIG. 2
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[1] Roederer and Wagler call attention to a black dotted appearance of these glands, "resembling a freshly-shaven beard." This is the *forme pointillé* of French writers. We have frequently seen this appearance, in persons dead of disease not affecting the intestines, produced by the exhibition of metallic salts. The cellular constituents of the intestinal glands become impregnated with the iron or copper salt, and, on contact with the bile, a black sulphide of the metal is formed, dyeing these minute corpuscular masses black.

In many places the follicles are seen to be dissected out, and only loosely connected with the surrounding shreddy tissue. The glands in the last foot of the ileum are always more or less implicated, and the innumerable and closely-placed solitary glands which form an almost continuous layer around the last two inches of the small intestine,—and which in some subjects are aggregated into one great terminal gland, the margin of which is coincident with the margin of the valve itself,—never altogether escape; and usually, indeed, the inflammation appears to have expended its whole force upon the glands of this part, and we find nearly the whole circumference of the last two inches of the mucous membrane greatly swollen, and in a ragged state of disintegration. The margin of the valve is not infrequently found as thick as the lips of the subject, and this part of the bowel usually presents a dirty ashy-grey appearance, veined with blackish-purple ramifications. Some glands are merely swollen, and their turgid, everted margins overlap the contiguous mucous membrane; others are converted into ashy sloughs (*forme gangréneuse*, Cruveilhier), often deeply stained with bile, sometimes dyed with blood. In some cases the ulcers are vascular and angry-looking; in others they are pale, anæmic, and have but slightly-raised margins. Just as the inflammation does not always equally affect all parts of the Peyer's patch, so we very often find that the ulceration may be partial. A given gland may present one or several distinct ulcers. They rarely exceed $\frac{3}{8}$ths of an inch in diameter; they have rounded, elevated borders, and at first sloughy, ragged, broken-down centres: the more advanced ones have the bare, smooth layer of circular muscular fibres, or only a little intervening areolar tissue, for their bases. In the early stage the muscular tissue is pale and free from inflammation, but sooner or later it becomes red, thickened, and soft, and soon yields to the ulcerative process. The longitudinal layer yielding in like manner, the diminishing base of the ulcer comes to lie upon the peritoneal coat. In proportion as the base of the ulcer now nears the peritoneum, so does that membrane increase in inflammation; and if the ulcers be deep and numerous, the inflamed patches become confluent, and the outer surface presents the appearance of intense inflammation, and is occasionally covered with a layer of plastic lymph. Occasionally the ulcerative process extends through the peritoneal covering, and symptoms of perforation ensue immediately, or are retarded for a time by the adhesion of solid lymph exuded upon its outer surface. The aperture formed in the peritoneum rarely exceeds three lines, and it is almost always formed within a distance of six inches from the ileo-cæcal valve. Sometimes the whole patch is converted by the confluence of smaller ulcers into a single deep ragged one, the sharp and perpendicular edges of which irregularly excavate the red, tumid mucous membrane immediately surrounding the diseased gland.

Occasionally the inflammatory process does not pass so soon into the gangrenous or ulcerative stage, and the glands become firmer and more prominent; the reticulations are completely effaced by the swelling, and the surface of these expanded, mushroom-like projections has a granular appearance (*forme granuleuse*, Cruveilhier). Glands in this condition may be restored to their natural state by resolution, or they may pass into the subsequent stages of gangrene or ulceration. The "*Plaques dures*" of Louis, which "à l'incision offrent une section ferme, lisse, et brillante," are very rarely observed in Enteric Fever distinguished from tuberculosis. In upwards of thirty fatal cases which I have examined, I have found this condition in only one (case 4), and in this I am inclined to attribute it to syphilitic taint. Fig. 9 represents one of the chancre-like Peyer's patches from case 4: *b* will serve to convey an idea of the uniform thickening of the gland, overlying the unaltered muscular and peritoneal layers.

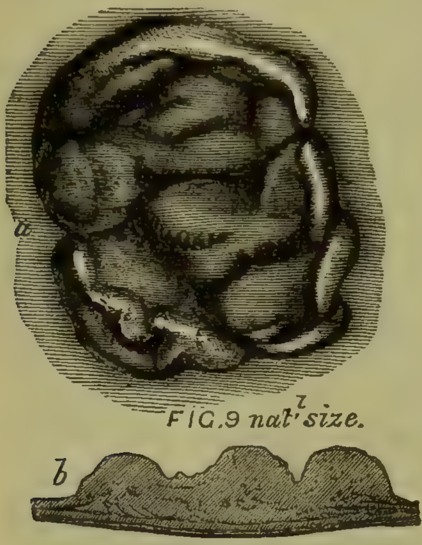


FIG. 9 natl size.

The solitary glands of the small intestine, and frequently also those of the cæcum and ascending colon, share more or less in the above-described changes. These minute glands occur in increased numbers towards the ileo-cæcal valve, where they become closely aggregated. Placed beneath the mucous membrane, and attached to its under surface, they lie loosely imbedded in the submucous areolar tissue, and in their healthy condition are hardly perceptible.

In many cases of Enteric Fever we find the last two feet of the ileum strewn with minute, round, semi-transparent elevations, varying in size from a mustard to a hemp seed. These are the solitary glands in a state of inflammation. In this early stage of the inflammatory process they have the appearance of a fine miliary eruption, and constitute the condition known as "*Psorenterie.*" When the solitary glands attain a larger size, and become a little harder and more opaque, the mucous membrane appears as if studded with pustules (*forme pustuleuse*, Cruveilhier). This appearance gave origin to the idea that Enteric Fever was "intestinal Variola." These swollen glands, however, are almost always solid: in only one case have I observed them to contain a yellow pultaceous matter, resembling inspissated pus.

If all the solitary glands be involved in the inflammatory process, the mucous membrane is thickly studded with them, and in the last two feet of the ileum the distance between them will average about $\frac{1}{8}$th of an inch. When an aggregation of a few solitary glands is swollen, a stud-shaped elevation is usually formed.

According to my own observations, the solitary glands are affected in proportion to the severity of the inflammation of the Peyerian glands.

In very rare cases the solitary glands alone are affected in Enteric Fever.

In many cases the disease is equally developed in the small intestine and cæcum; once I have seen death from perforation of the cæcum. Occasionally the large intestine is more extensively ulcerated than the small. In case 19, for example, the small intestine escaped, and the inflammation affected the solitary glands of the large intestine almost exclusively.

In proportion as the solitary glands are inflamed and swollen, they cause a projection and thinning of the mucous membrane. Attentively examined under water with a pocket-lens, they are seen to be of a delicate pink colour, and exhibit a minute dark central point. Occasionally the swollen gland presents a yellowish summit surrounded by a minutely injected areola of converging blood-vessels. Ulceration commences by the softening and abrasion of the mucous membrane around the summit of the gland, the disintegration then becomes deeper, and spreading outwards, minute circular ulcers, with sloughy, shreddy centres, and purple, tumid margins, are formed. These ulcers rarely exceed $\frac{3}{8}$ths of an inch in diameter. Their further progress is identical with that of the ulcerated agminated glands, and they are equally liable to produce hæmorrhage and to perforate the bowel. In most cases we find a few of the solitary glands of the cæcum and large intestine thus inflamed and ulcerated. Occasionally the glands of the large intestine are more or less implicated along the chief part of its extent, and by the confluence of the small ulcers very large ones are sometimes formed in the cæcum and ascending colon. The direction of these ulcers is generally transverse. In Enteric Fever, ulceration always commences in the solitary or agminated glands; and if these were the only "follicular glands" in the intestinal canal, the term "Follicular Enteritis," by which Enteric Fever has been distinguished, would be a very suitable one.

We have now to consider the *nature* of that morbid process, the effects of which have been described. From the description just given, it is clear that the process is an inflammatory one. Usually there is evidence of very acute inflammation. It will be inferred from the foregoing description of the diseased glands that the inflammatory products are formed around the closed follicles, and not in their interior. Very careful observation leads me to speak positively on this point. If the new material were formed within the closed follicles, as Goodsir concludes, the follicles would indeed "become much distended," and, as a result, they would form projections upon the surface of the Peyer's patch, which I have never observed to be the case. On the contrary, I have always found them in the earliest stages of the inflammation to be placed far below the swollen ridges of mucous membrane and submucous tissue surrounding them; and in the latter stages, the follicles are completely buried beneath the inflamed surface of the patch, and concealed from view, and it is only when the excessively vascular and turgid ridges of the mucous membrane and subjacent tissue are disintegrated, that the follicles are again discovered, lying deeply in the abundant submucous tissue, and exhibiting little or no increase of size. The parts immediately surrounding them appear to have undergone considerable disorganization; for the follicles are often dissected from the surrounding parts, and remain attached to them by only a few tough fibres. In health, each follicle is surrounded by a close network of blood-vessels, which, as far as I have observed, chiefly constitutes the wall of the little gland; from this parietal network other branches, exceedingly fine and delicate, pass towards the centre of the parenchyma. If the vascular excitement be moderate, the central, as well as the circumferential parts of the gland, may increase in size: but usually the inflammation is acute. Cut off from all other parts of the circulation, and surrounded by inflamed vessels, congestion and stasis would very soon occur in the delicate vessels which pervade the parenchyma; and thus, whilst the parts external to the follicles would be increasing under the influence of the inflammation, the central parenchymatous parts would undergo no increase, but would tend to atrophy and disintegration. Hence the formation of the centrifugal ulcers and sloughs around the follicle; and such indeed must always be the results of inflammation in parts which have a similar arrangement of blood-vessels within them.

Structure and Characters of the Inflammatory Product.—This we find to be cellular. On examining vertical sections of Peyer's patches in the early stage of inflammation, represented in fig. 1, we find that the submucous tissue is composed of a very loose network of very elegantly waved and reticulated fibrous tissue, from which the so-called walls of the closed follicles are not defined. The meshes of this network are filled with finely granular corpuscles of various sizes, chiefly spherical, and averaging $\frac{1}{3000}$th of an inch in diameter. (Fig. 4.) A few cells of adipose tissue, arranged in single rows, are occasionally seen. Sections through the more advanced and ulcerated patches present the same arrangement of the fibrous stroma; the cells are equally numerous, but they are a little larger, and of more uniform diameter, averaging $\frac{1}{2100}$th of an inch, and a little more darkly granular. (Fig. 5.) Here and there a corpuscle is observed containing one or more spherules of oil. Sections of the firmer swellings (*forme gaufrée*), and of those in a more advanced stage of ulceration, show that the corpuscles undergo fatty degeneration, and subsequent molecular disintegration. In these we observe multitudes of enlarged corpuscles containing spherules of oil, and much intercorpuscular molecular matter. (Fig. 6.) Rokitansky speaks of "the deposition of a typhous product" in the inflamed glands. The swelling, according to my own observation, is due to the rapid growth of the corpuscles

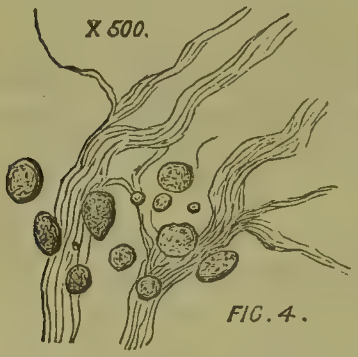
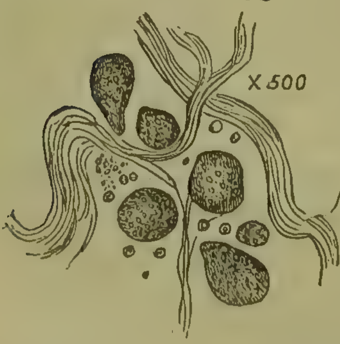


FIG. 4.

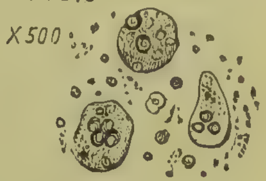
FIG. 5



forming the parenchyma of the glands, whether Peyerian or mesenteric.

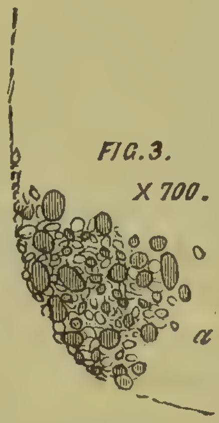
Not infrequently fibrinous exudation forms upon the surface of the ulcerated gland (case 5), or amongst its cellular constituents (case 4). Sections of the gland, which I have delineated in fig. 9, showed the elements represented in fig. 6, interspersed with minuter corpuscular matter and molecular fibres.

FIG. 6



The *villi* upon the diseased patches and contiguous mucous membrane have a smooth outline and are denuded of their epithelium. They present a finely granular appearance, due to the presence of innumerable homogeneous, yellowish-tinted, refractive granules, which average $\frac{1}{5000}$th of an inch in diameter. Some attain $\frac{1}{2500}$th of an inch; others are mere molecules. Fig. 3 represents a minute portion of such a villus highly magnified.

FIG. 3.



Stages of the Local Disease.—Since the disease is usually developed so very insidiously, it will be difficult, and in the early stages impossible, to predicate with certainty the actual condition of the intestinal glands. The following generalizations, however, may prove useful (see also Diagnosis). For the first *nine* days the glands are undergoing inflammatory swelling, and at the end of this period they will be found projecting three or four lines from the mucous membrane, in the form of red, or purplish, fungous, soft excrescences, free from erosion. If death occur any day before this period, we shall find the glands more or less advanced towards this condition. About the *tenth* day the inflammation either subsides or increases. Resolution is effected in the usual way by diminution of the vascularity and swelling. If the inflammation increase, the swollen glands become a little firmer, and on the *eleventh* and *twelfth* days present softening, and erosion of the mucous membrane covering them. *Fourteenth* day: circular disintegrations around the follicles; a spongy, sloughy appearance of the abraded patch, which is frequently stained of a deep ochre colour by the bile—the formation and separation of ashy sloughs. *Sixteenth* day: complete separation of the sloughs, leaving ulcers limited below by muscular fibres or peritoneum, and surrounded by red, swollen margins of mucous membrane; erosion of blood-vessels, and hæmorrhage. *Twentieth* day: cicatrization begins. *Fortieth* day: cicatrization completed.

Reparation of the Intestinal and other Lesions.—In those who have died during a relapse of Enteric Fever, or at an advanced period, of pulmonary or other complication, we may often observe the process of reparation of the local disease. The following case exhibits the condition of the abdominal viscera during recovery from a severe attack of Enteric Fever, with pneumonia. The patient died of gangrene of the cheek (cancrum oris) and lungs.

Case 6.—Joseph Taylor, aged 15, came under my care, August 15,

1865. He had been ill *three* days with headache, nausea, diarrhœa, and fever, and presented on admission all the symptoms of well-developed Enteric Fever (without rose rash, which never appeared), and pneumonia of the left lung. On the *sixteenth* day: pulse 144; respirations reduced to 28; diarrhœa and abdominal tenderness somewhat abated; dulness and fine crepitation over both bases of lungs behind. Three black sloughs, the size of peas, have formed in the mouth, two on the gums and the third on the centre of the left cheek. *Nineteenth* day: pulse 162, hardly perceptible; tongue dry and brown; bowels very loose; passed a considerable quantity of blood in the stools to-day; slough on the cheek spreading; cheek hard, and swollen. *Twenty-second* day: pulse 144; moderate intestinal hæmorrhage every day; diarrhœa restrained; cheek much swollen, duskily flushed, hard, and shining; respirations less frequent. *Twenty-fourth* day: bowels quieter; no more hæmorrhage; takes drink well and sleeps fairly; slough of cheek extending, those of the gums separated with the loss of two molar teeth. *Twenty-sixth* day: remains quite conscious and takes drink well. The left cheek is livid externally, and the eyelid closed by the swelling. From this date the pulmonary and abdominal symptoms declined, and the bowels acted naturally, the stools becoming solid. The gangrene, however, spread externally, and involved all the central parts of the cheek in a large circular slough, and the patient gradually sank, retaining a clear intellect throughout the disease, and died on the *thirty-second* day.

Autopsy.—Body much emaciated. *Chest*—lungs weighed together twenty-four ounces; apex of the left gangrenous, and partially broken down; lower lobes of both firm, slightly crepitant, pale-red, friable—recovering from pneumonia—here and there a small circular ashy slough; no trace of tubercle. Heart healthy; blood fluid; right internal iliac vein, at its junction with the cava, firmly plugged with a yellow, friable clot. *Abdomen*—liver weighed two pounds six ounces; firm; lobules indistinct, with a whitish speckling in the form of minute stellæ; the gland did not appear to me to be fatty, but microscopic examination showed the cells to be greatly enlarged, destitute of pigmentary matter, and replete with oil. Bile abundant, pale ochre-coloured, watery, acid. Excepting a few patches of minute injection of the mucous membrane of the stomach, the alimentary canal was healthy to within four feet of the ileo-cæcal valve. This lower portion of the ileum was much injected and dark red. At four feet from the valve, a small Peyer's gland, the lower end of which presented a round, gently elevated swelling, with a central irregular excavation the size of a hemp seed, limited externally by the healing, granular margin of the pink mucous membrane. Three inches lower down, a larger gland, the lower half healthy, the upper with four cicatrizing ulcers—three so far healed as to be converted into minute stellate chinks, surrounded by pale red, wide, smooth borders, scarcely elevated above the surface of the healthy portion of the gland. Below this gland were nineteen minute cicatrizing ulcers, chiefly of the solitary glands, all with rounded, smooth, very soft vascular borders firmly attached to the less vascular transverse or longitudinal layers of muscular fibres, which formed clean, smooth bases to all the ulcers. Next occurred six large ulcers caused by the destruction of the whole of the large Peyer's glands of this part; they formed large, smooth, and soft, interrupted depressions, limited below by the very distinct reddish-grey muscular fibres, and surrounded by pale-red, raised, and rounded sinuous borders reposing upon the muscular layer: two or three of these ulcers presented rounded islets, or projections of smooth red, mucous membrane running in from the raised border of the ulcer, and on a level with it. (Fig. 10.) One of these large patches presented a minute contracting ulcer at either end, the intervening space being occupied by a smooth, greyish-white, opaquish, slightly-depressed membrane. Nearer the ileo-cæcal valve were thirty-one other ulcers chiefly affecting the solitary glands, and varying in dimensions from mere linear chinks to the three-fourths of an inch. All were in process of cicatrization. In the next portion of the ileum—the last four inches—there were a great many similar ulcers, all clean and healing, but not quite so far advanced in this process as those situated higher up. There were three small and distinct healing ulcers in the colon, the last one situated at the distance of a foot from the cæcum. The cæcum, and rest of the large intestine, including the rectum, were perfectly healthy. The solitary glands were all visible and marked by a central black dot just as they appear in the meconium-stained bowel of a newly-born infant.

The mesenteric glands were, for the most part, as large as almonds, and so flaccid that they could scarcely be distinguished, in the mesentery, between the thumb and finger: they were of a dusky-grey or ashy colour, and of an almost leathery toughness. Entire sections of them could be readily made, and these were as tough as fibrous membrane, and presented an abundant, finely fibrous stroma, the ordinary corpuscles, and a considerable quantity of highly refractive granules.

The receptaculum chyli and thoracic duct were collapsed and empty. The spleen weighed five ounces and a drachm; it was of natural consistence, and presented a bright reddish-brown colour on section. The remaining viscera were apparently healthy.

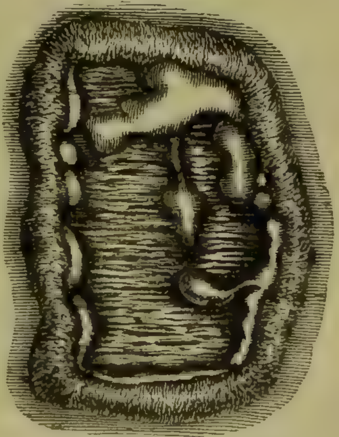


FIG. 10 nat size.

Floated under water, the rounded vascular borders of the healing ulcers present a double margin, the villi are seen to terminate in a wavy line, and from within and below the border so formed projects the paler and quite smooth soft border of advancing granulations. (Fig. 10.) Some of these spring up from the base of the ulcer, and form islands, which ultimately become confluent with each other and the margins of the ulcer, to form a smooth, depressed membrane, which always remains destitute of villi and of closed follicles. In some of the cicatrized ulcers we occasionally observe a little cluster of closed follicles, but this simply points to the fact that a portion of the closed follicles of that particular gland escaped injury. After these follicles have been removed in the inflammatory process they are never regenerated. Years after an attack of Enteric Fever the ulcerated Peyer's patches will be found to be replaced by pale, smooth, slightly-depressed, but unwrinkled membranes, which are more firmly adherent to the muscular layer than the healthy gland, and remain permanently destitute of villi.

(*b*) *The Mesenteric and Mesocolic Glands.*—Just as inflammation of the tonsils induces vascular excitement and swelling of the lymphatic glands, situated about the angle of the jaw, so does inflammation of the solitary and agminate glands excite inflammation in the corresponding glands of the peritoneal folds. The swelling of the latter is always proportionate to the degree of the intestinal irritation; the glands, therefore, which lie in the angle of junction between the small and large intestine are those most affected. In every case of Enteric Fever we find that the mesenteric glands are more or less congested, swollen, and softened. They are usually of a dark purple colour, and of the size of hazel-nuts. Some often attain the size of a walnut. Bisected with a sharp scalpel, the outer portions are seen to be of a uniform dark purple colour, the central parts are less vascular, and the yellowish-white parenchyma is veined with diffuse purple streaks, and a mottled appearance thus produced. The parenchyma seems yellower than usual, but this is simply the effect of contrast. The gland is so soft that it is difficult to make a thin section of any extent. Microscopically examined, we find it to be composed of an exceedingly delicate, friable, scarce stroma of indistinct fibres, and of molecular corpuscles of various sizes. These latter constitute nearly the entire gland; they are for the most part spherical and nucleated: the most numerous average $\frac{1}{3600}$th of an inch in diameter; the larger present well-formed nuclei, and average $\frac{1}{2300}$th of an inch in diameter. (Fig. 7.)

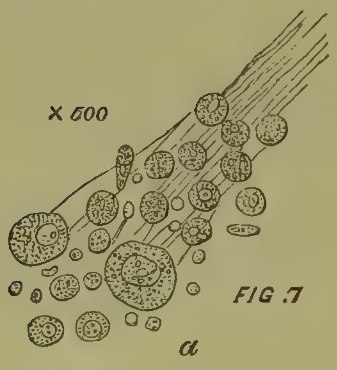


FIG. 7

In the subsequent progress of the disease the glands may return to their normal condition, or the cells may break down to a creamy fluid. In one or two cases this puriform fluid has increased to such an extent, as to rupture the peritoneal covering of the gland, and general peritonitis has followed the extravasation of its contents.

As soon as resolution of the inflamed, and cicatrization of the ulcerated glands of the intestine have taken place, the mesenteric glands begin to decrease, and become for a time shrunken, flabby, and tough.

In those cases in which I have made the necessary examination, I have found the receptaculum chyli and thoracic duct empty and collapsed.

(*c*) *The Spleen* is severely congested in almost every case—probably, during the inflammatory period of the disease, in every case. It is usually enlarged to twice or thrice its natural size; occasionally it is found four or five times larger and heavier. Its colour is uniformly purplish-black throughout, and it is so soft and friable that it may be reduced to a semifluid pulp with the greatest ease. Minute granular corpuscles, fibre-cells, and molecular branched fibres are the only structures I have been able to detect under the higher powers.

(*d*) *The Liver.*—A morbid condition of this organ and its secretion has been very generally observed. Forget does not specially mention the condition of the liver in many of his cases. Of others he records the following observations: "Liver normal, gall-bladder containing much, or little, thin bile." (Obs. xlv. xlviii. lv.) "Liver voluminous, possessing a fatty appearance." (Obs. lx.) Liver voluminous, gall-bladder almost empty." (Obs. lxviii.) "Liver presented a little softening in its right lobe; the gall-bladder contained a thin bile, slightly coloured, like water." (Obs. lxxiii.) The liver was softer than natural in thirty-two out of seventy-three cases examined by Louis, Jenner, and Murchison. (Murchison, p. 555.)

Louis states that the *volume* of the gland was augmented in $\frac{1}{10}$th of his cases, and in these it had lost its consistence; the *consistence* was diminished, the tissue of the organ being sometimes soft, sometimes friable, in the majority of his cases, and in none did it appear to him to be firmer than natural; *softening* existed in nearly half the cases, and in four to such a degree that the fingers sunk into the gland substance without resistance; the *colour* was natural in only twelve of the subjects examined by him; it was redder than usual in eight, five of which were examples, more or less marked, of sanguineous engorgement. This appearance was noticed a little more frequently in those who died at an early period—from the eighth to the twentieth day. The *bile* was sometimes red, and very fluid in different degrees in about half the cases; in ten, it was more abundant than usual. (Louis, Rech. Fièvre Typhoïde, vol. i. p. 269 et seq.) Another careful observer, Grossheim, remarked that, in all the cases observed by him, "the liver never retained its normal colour, and the bile was always much thinner and clearer than in the healthy state. It was frequently transparent, sometimes clear yellow, sometimes of a dirty whitish colour; in quantity, it was sometimes normal, rarely increased, but most frequently of all it was so diminished that scarcely any was left." (Edinburgh Med. and Surg. Journal, 1837, vol. xlviii. p. 178.) Stannius examined twenty-three fatal cases of Enteric Fever. "In the majority, the liver appeared to be of normal consistence and colour; not infrequently it was softened generally or partially. Almost always, both in those cut off at the height of the disease, and in those destroyed at later stages, the gall-bladder contained pale yellow, or yellowish-green, often watery mucous fluid, not reddening litmus paper nor tinging the skin." (Ibid. p. 174.)

My own observations agree with the foregoing; but as to the frequency with which the liver is found in a morbid condition, I am led to conclude that the gland *never* escapes without some alteration in its texture. In every case which I have examined, I have found the liver in a more or less advanced state of fatty degeneration, and in almost every case noted an increase of weight. Even when the gland is of normal size and to all appearance healthy, or only a little pale, microscopic examination will show very considerable degeneration of the hepatic cells. In case 1, above recorded, the liver cells were greatly enlarged, averaging $\frac{1}{1000}$th of an inch in diameter, and frequently containing spherules of oil $\frac{1}{2000}$th of an inch in diameter. The bile in this case had the low specific gravity of 1018 and strongly reddened blue litmus paper. After depositing an abundant pale ochre-coloured granular-looking matter, composed of columnar epithelium, it had the colour of whey, or pale urine with a faint greenish tinge. I have constantly found the bile thin, watery, and easily filterable ; in one case the specific gravity was as low as 1011·2. Filtered, and evaporated on a water bath, such altered bile yields only a small quantity of black solid matter, greenish-brown, by transmitted light, and wholly soluble in water. The bile itself, or this solution, gives slowly and faintly, sometimes imperfectly, the characteristic reactions of bile when tested with the mineral acids, or Pettenkofer's test. The bile has a strong post-mortem odour, and in one case which I examined twelve hours after death, when the viscera were still warm, and the blood steamed on exposure to the frosty air, it smelt strongly of sulphuretted hydrogen.

The morbid changes, above described as affecting the intestines, the mesenteric glands, the spleen, and the liver, are the constant and essential lesions of Enteric Fever. We now pass on to a cursory examination of such morbid phenomena as are exhibited by the other organs of the body.

Tongue.—The general condition of this organ has been described. The characteristic features are, unusual redness of its edges, with enlargement and prominency of the fungiform papillæ, in the early period of the disease ; and a wrinkling and cracking of the dry glazed surface, with contraction and reddening of the whole organ, at a later period. The cracks are very painful and often bleed. If the tongue remain moist, it is usually flabby, indented, and covered with white fur. In this condition it occasionally presents spreading ashy ulcers upon the tip and sides ; and sometimes deep fissural ulcers, with pale everted margins, form across the dorsum. When nervous symptoms predominate, the tongue becomes covered with a thick, brown, firmly-adherent crust, very dry and hard, and reticulately fissured.

In several cases, I have observed great congestion and swelling of the follicular glands at the base of the organ.

The Lips and orifices of the *nostrils* are often cracked and inclined to bleed.

The Tonsils are rarely affected ; abscesses have been observed in them in a few cases.

The Pharynx and Œsophagus.—Louis found small round or oval ulcers of the mucous membrane of the lower portions of the pharynx and œsophagus in about a sixth of his cases.

The Stomach, Duodenum, and Jejunum are usually healthy. In some cases they present morbid conditions, such as softening and minute ulcerations of the mucous membrane, which are common to all inflammatory diseases.

The Pancreas.—I have usually found this gland harder and with the lobules more distinct than in health, as if shrunken. Otherwise it has appeared healthy.

The Urinary and Generative Organs are in the normal condition, or only slightly congested.

The Epiglottis, Larynx, and Trachea are occasionally ulcerated. The mucous membrane of the bronchial tubes is usually red and swollen.

The Lungs present in almost every case evidence of pre-existing inflammation. (See Associated Pathology of Enteric Fever.)

The Muscular System.—Agreeably with what is observed in other protracted diseases of an acute character, the muscular tissue is found to be liable to degeneration in Enteric Fever. Zenker (Veranderungen der Muskeln in Abdominal. Typhus, 1864) describes two forms of muscular degeneration—granular and waxy. The granular form consists in the deposition of minute highly refracting granules in the contractile tissue, giving to the fibres a dark appearance by transmitted light and obscuring the striæ. This molecular deposit is not wholly composed of fat. The degenerated fibres are very friable. The waxy form consists in the transformation of the sarcous tissue into a homogeneous colourless mass, glittering like wax and causing a complete obliteration of the striæ and nuclei of the fibres, the sarcolemma remaining intact. The waxy cylinders, thus formed, crack up into numerous fragments, which crumble down into a finely granular detritus, and this is gradually absorbed. The muscles most liable to degeneration are the adductors of the thigh and the abdominal recti. The affected muscles are of a pale greyish-red colour. Rokitansky observed rupture of the abdominal rectus in Enteric Fever, and attributed it to spasm. Virchow noticed rupture of the muscles associated with friability of the muscular fibres in four cases of Enteric Fever. Zenker noted eleven such cases, all of which occurred in Enteric Fever. The rupture occurred most frequently, but by no means exclusively, in the rectus abdominis, transversalis abdominis, pectoralis minor, triceps brachii, and psoas. The author last mentioned attributes the rupture of the muscles, and extravasation of blood into their substance, to the degeneration of the fibres above described. The rupture tends to produce hæmorrhage, and this leads to the formation of collections of sanies or pus, which must be distinguished from general pyæmic deposits. Abscesses in the muscles are very rare in Enteric Fever.

The Skin presents us with one of the characteristic symptoms of Enteric Fever, the "*tâches roses lenticulaires*" of Louis. These spots closely resemble the papules of variola during the first few hours of their existence, but they are not quite so large nor so hard. They form slight, rounded, discrete elevations of a pale rose colour, which fades away at the base, forming a moderately distinct circular outline. Each rose papule is a minute circumscribed inflammatory centre, from which the blush disappears on pressure. These spots usually appear on the abdomen and chest alone, but they are often found on the back. They are seen occasionally on the face and upper and lower extremities. The eruption is not always present. "Of 1820 cases admitted into the London Fever Hospital during ten years, it was noted in all but 224, or 12·3 per cent." (Murchison, p. 470.) The rash usually appears on the supervention of the acute febrile symptoms. It may be looked for at the end of the first week, and may continue as long as the febrile symptoms and diarrhœa persist. The total number of spots rarely exceeds fifty; in some cases they are innumerable. There is no relation between the quantity of the rash and the severity of the symptoms. It appears in successive crops; at first only two or three spots may be observed, next day four or five fresh ones, the next as many more. Each crop persists for a few days and then disappears. According to Barthez and Rilliet, and Murchison, the spots are fewer in children than in adults; and the former two observers state that in the same class of patients they are oftener absent in the severe cases than in the mild. These rose spots occasionally appear in other acute diseases. In a severe case of typhus in a powerful fair-complexioned man I noted a very copious eruption of rose papules upon the chest and abdomen; they preceded the typhus rash, and had wholly disappeared when this became petechial.

The departure of the fever and the re-establishment of the cutaneous function is often announced by the eruption of *sudamina* over the whole of the chest and abdomen.

Roughness and minute desquamation of the cuticle, especially of that covering the abdomen, are observed after the cessation of febrile symptoms in severe cases. The desquamation occurs independently of the pre-existence of sudamina, which alone is sufficient to produce it.

The temperature of the skin usually undergoes a progressive increase during the first fourteen days of the disease, attaining, in severe cases, 104°, subject to the morning and evening vacillations, which are observable in other febrile conditions. If the abdominal or pulmonary symptoms undergo no amelioration, this temperature is often maintained during the earlier part of the day. When the intestinal inflammation proceeds to extensive ulceration, this high temperature may persist more or less continuously for weeks; but usually during the third week there are peculiar alternations of temperature, ranging from 4° to 6° per diem, the higher readings being observed during the hectic exacerbations which take place in the evening. Recovery in such cases is attended by a gradual diminution of temperature. In more favourable cases the resolution of the inflammation is declared by sudden falls of temperature.

When the fever is prolonged, the pungently hot skin becomes very harsh, and the papillæ as prominent as in the *cutis anserina.*

The Lymphatic Glands are usually only secondarily affected in cases complicated with ulceration of the pharynx and erysipelas of the surface. In young children, suppuration of the cervical glands about the angle of the lower jaw is not very uncommon: three such cases have lately come under my care. Parotid inflammation, which is so common in typhus and in scarlatina, is rare in Enteric Fever.

Nervous System.—The only lesions discoverable are slight subarachnoid effusions, fulness of the blood-vessels, and slightly increased vascularity of the cerebral substance.

Circulatory Organs.—In protracted cases the muscular tissue is liable to fatty degeneration, and this change becomes first apparent in the left ventricle of the heart.

The Blood.—M. Trousseau, in speaking of intestinal hæmorrhage in Enteric Fever, says the blood is exhaled by the mucous surface, as occurs in hæmatemesis, epistaxis, &c. "The proximate cause of this sanguineous exhalation," he goes on to say, "is a profound alteration experienced by the blood, which is found in that state which one has termed 'the state of dissolution.'" (Clin. Med. p. 230.) M. Forget examined 123 specimens of blood, derived from patients in all stages of Enteric Fever. Of the blood drawn during the first period of the disease, only about $\frac{1}{18}$th of the specimens presented appreciable softening. In the second period $\frac{1}{7}$th of the specimens exhibited this change.

He concludes generally that an appreciable alteration of the blood in the several periods of Enteric Fever cannot be accepted as a general fact; that the blood is rarely altered in the first period; that the alteration is more marked in proportion as the disease is more advanced; that the alteration is not always in proportion to the gravity of the disease. (Forget, Sur l'Etat du Sang dans l'Entérite folliculeuse: Gaz. Médicale.)

My own observations of the condition of the blood of those who have died from Enteric Fever accord with those of M. Forget. In subjects dead in the third week of the disease, I have frequently found firm colourless clots of fibrin in the heart and roots of the great vessels. In protracted cases the blood not only becomes very thin, but is also much diminished in quantity, from sheer inanition.

PATHOLOGY.

If we carefully regard the incipient symptoms of Enteric Fever, we shall find that they have reference to derangement of the hepatic function. Often, long before the graver symptoms are developed, the patient loses appetite, the bowels are constipated, and the stools pale; the tongue is foul, and the digestion much impaired. All these symptoms point to a defective secretion of bile, and to a state of approaching inanition. Such a torpid condition of the liver may be produced in two ways in the development of Enteric Fever. It may result from severe or prolonged vascular congestion, in which the other internal organs participate; or it may be the effect of some morbific agent, carried by the portal vein from the intestinal surface into the liver, and causing, by a direct action upon its secreting corpuscles, derangement, or more or less complete paralysis, of its functions.

If in any case a poison be not decomposed in its passage through the alimentary mucous membrane, it must of necessity be admitted into the liver. We know how readily mineral poisons are conveyed and arrested there, and we recognise the effects of certain vegetable substances upon the hepatic secretion. From these facts, and from its situation between the intestinal and general circulations, we may reasonably conclude, that it is one of the offices of the liver to arrest noxious matters in their way from the portal into the general circulation, to neutralize or decompose them, or to eliminate them from the blood, and throw them out again through the bile ducts into the intestine.

The very admission of deleterious agents into the portal circulation must lead, by diminishing the reciprocal attractions of the portal blood and the hepatic corpuscles, to congestion of the whole portal circulation.

Thus prepared, and by that concurrence of related actions which we everywhere witness in the body, the congested capillaries of the intestinal mucous membrane relieve themselves by a copious watery exudation, by means of which the poison set free by the liver is washed out of the alimentary canal. Such probably is the mode of action of elaterium, colchicum, &c. But it is the special function of the liver to prevent putrid decomposition within the body. If therefore the function of this gland be depressed, as in a case of simple vascular congestion from exposure to cold, for example, a septic poison may be generated within the body, and set up all the symptoms which follow the introduction of a similar poison from without. Doubtless, so long as the liver is in an active healthy condition, any septic poison taken into the alimentary canal would generally be neutralized; but if the gland should happen to be torpid at the time, then the unaltered poison, upon admission into the liver, would possibly arrest the secreting corpuscles in the elimination of that very fluid which has the power of rendering it innocuous. Very little is known of the derangements to which the liver is liable, and of the alterations which its secretion undergoes. We readily obtain evidence of the grosser irregularities of the kidneys, but we can judge of those affecting the liver only by the colour of the fæces—a good general guide, no doubt; but only rarely is this means of diagnosis available in the incipient stage of diseases.

Primary vascular congestion of the liver, no matter how produced, leads to a vitiation of the secretions of the alimentary canal; nervous exhaustion results from arrested nutrition. Under these conditions the liver begins to degenerate, and the intestinal mucous membrane tends to ulcerate, the blood is imperfectly depurated, and general febrile disturbance ensues. Surely if high fever, violent delirium, and coma are the consequences of acute suppression of the bile, the pyrexia, headache, and the most severe delirium, which ever accompany Enteric Fever, may be fairly attributed to that diminution and derangement of the hepatic function which invariably accompany this disease.

That the liver is early and gravely deranged in Enteric Fever is proved by the facts already mentioned in the morbid anatomy of the disease, and by the prominency of those symptoms which have led observers in all ages to designate it by the terms, "bilious, gastro-bilious," &c.

In place of a thick, heavy, alkaline secretion, rich in biliary acids and colouring matter, we find a watery, neutral, or often excessively acid bile, notably deficient in its essential constituents, and sometimes putrid at its very source. M. Trousseau considers the flux from the bowels to be of the nature of a specific catarrh. But what is the *nature* of this specific catarrh? Is the bowel endeavouring to supply defective action of the liver by carrying away, in some unformed state, constituents of the blood which that gland should have removed as glycocholic and taurocholic acids? We do not think such a theory necessary. At the commencement of the disease there is probably some attempt at elimination, but in the subsequent stages we believe that the diarrhœa and intestinal lesions are rather due to congestion and mere local irritation than to any specific cause. This would appear to be the case from consideration of the fact that if we restrain the diarrhœa—the assumed means of elimination—we do not aggravate the general symptoms, but positively ameliorate them; and in most cases marked improvement follows the complete arrest of the diarrhœa.

Not the least important function of the liver is to prevent by its antiseptic properties the decomposition of the chyme; take away this preservative influence altogether from the system, and fermentation with the escape of gas and tympanitic distension follow. The impure chyme irritates the debilitated and congested mucous membrane, and what wonder then if inflammation, ending in ulceration of Peyer's patches and the follicular glands, should result?

But why should these particular structures suffer more than any other parts of the intestinal canal? For two reasons, we think: first, on account of the greater vascularity of these glands, whereby they most readily participate in local congestion, and, as has been shown, the arrangement of blood-vessels within them, which, when the circulation is obstructed, renders them liable to sloughing; and secondly, on account of their delicate cellular structure, for in febrile conditions it is the active growing corpuscles of the parenchymatous organs which most readily participate in the inflammatory process.

That the glands of the lower three feet of the ileum should be most affected may perhaps be regarded as a significant fact, and it is one for which it is difficult to find a satisfactory explanation. Anatomy will not allow us to ascribe a difference in function between the solitary and agminated glands lying near the junction of the small and large intestines and those removed to a greater distance from it; nor do we find that the glands of the upper and lower parts of the ileum have such a difference in their immediate associations as would account for unequal participation in general disease; and we should, therefore, be led to assume that if the solitary and Peyerian glands were employed in some general process connected with the elimination of a blood poison, they would be similarly affected. Such, however, is rarely or almost never the case in Enteric Fever, for the Peyerian glands of the lower third of the ileum are almost always found in a state of extreme inflammation when those of the upper two-thirds exhibit no morbid change, and we never find Peyer's glands of the upper portion of the ileum ulcerated when those of the lower are uninflamed.

The following considerations may afford some explanation of these facts. *First:* there appears to be a greater tendency to congestion of the lower than of any other portion of the ileum, due to the greater number of vascular solitary and agminated glands situated there, and also to the manner in which the small and large intestines are united. The abrupt fold forming the ileo-cæcal valve is similarly constituted to the anal sphincter, and, like it, necessarily causes some arrest in the flow of blood beyond its margins. We recognise, therefore, a predisposition in the lower part of the ileum, to participate in inflammatory action. *Secondly:* if we now regard the derangement which exists within the digestive canal, we may be able to find an exciting cause in the altered action, which doubtless results from disturbance of the reciprocal action of parts engaged in the same function, but separated from each other by a considerable distance. Can we, for example, attribute the lesion of Peyer's glands in the lower portion of the ileum to defective action of the glandular apparatus situated in the higher portions of the alimentary canal? The liver, we have found, secretes bile deficient in those essential constituents which exert an important influence upon the digestive process. The defective bile probably contains sufficient of these constituents to maintain healthy action in the upper portion of the small intestine, but becoming exhausted of these in the lower, it there fails to exercise any antiseptic influence, and of itself induces unhealthy action.

But, it may be argued, if this were the true explanation of the lesions of the small intestine, how is it that the large bowel escapes; for, according to the theory, we should expect to find that the intestinal lesions would progressively increase from the lower third of the ileum downwards, instead of being confined, as is usually the case, to the lower third of the ileum and cæcum? The frequent immunity of the large intestine from any considerable participation in the disease may be explained by supposing that the irritation set up in the lower portion of the ileum, by the vitiated bile, causes such a copious exudation of fluid from this part of the alimentary canal, that the irritating matter is diluted, and at the same time so rapidly carried away through the great intestine, that the lower portion of the alimentary canal usually escapes any severe implication in the intestinal lesion.

There can be very little doubt that the dejections in Enteric Fever are chiefly thrown off from that part of the intestinal canal where the inflammatory irritation is greatest—viz. the lower portion of the ileum. The cæcum, where the secretions are necessarily retained for a time, is often as gravely affected as the last six inches of the ileum. In some cases, moreover, the large intestine is often severely involved in the disease, and occasionally, as we shall have an opportunity of showing, it is exclusively affected,—a fact quite consistent with the theory here advanced. *Thirdly:* the localization of the intestinal disease may be supposed to arise from derangement of that particular part of the sympathetic nervous system, which is distributed to the lower portion of the ileum, just as destruction of the eyeball may follow injury of the orbital branches of the fifth nerve. Morbid anatomy fails, however, to reveal such derangement of the sympathetic plexuses; and if it did, there would still remain the difficulty of accounting for a general febrile condition in such limited defect of nervous action. The question naturally arises to every inquirer, whether the symptoms of Enteric Fever are to be attributed to general blood-poisoning, or whether they secondarily arise as a consequence of a localized intestinal lesion. From the foregoing observations it will be seen that we are induced to conclude that the disease arises from a vitiation of only a portion of the venous blood, and that the constitutional symptoms are in many cases due to consequent derangement of the hepatic function. If we accept this view, we shall be at no loss to account for the great variation in the *nervous symptoms* observable in this disease. Some patients retain a clear intellect to the last hour of their lives (*e.g.* cases 5 and 6); others lapse into a state of stupor or coma at a very early period of the disease (*e.g.* case 3); and the majority manifest great nervous irritability and prostration, and, at some period or other, more or less delirium. In every case there can be no doubt that the derangement of the digestive, cutaneous, and pulmonary functions results in an impure condition of the blood; but we consider that in many cases the nervous symptoms are due to nervous exhaustion from inanition, or to active meningeal congestion, rather than to a specific blood-poisoning. The delirium partakes very much of the character of delirium tremens, and there is frequently very notable vascular excitement of the cerebral circulation. In those cases in which the cerebral symptoms are predominant, we are forced to recognise a general blood-poisoning, and then the question arises, Is this due to more or less complete suppression of the hepatic function, or to the admission of a specific poison into the general circulation? Probably it may be due to both of these causes. If the poison be arrested by and thrown out from the liver, no general blood-poisoning, and, therefore, no grave nervous symptoms, may ensue. If the liver be unequal to the arrest and elimination of the poison, it passes unaltered from the portal into the general circulation, and symptoms of general blood-poisoning at once appear; and if the gland be so far deranged in the process of elimination as to become almost paralysed in its function, more or less complete suppression of bile would be an additional cause of the cerebral symptoms.

ASSOCIATED PATHOLOGY OF ENTERIC FEVER.

Pneumonia.—The lungs and the intestinal and mesenteric glands manifest very great sympathy in morbid action. In two, at least, out of every three of the many cases of pulmonary phthisis which I have examined, I have found the solitary and agminated glands of the lower portion of the ileum and the mesenteric glands more or less infiltrated with tubercle, and the former often very gravely ulcerated. The same sympathy is observed when the lungs are the seat of common inflammation, and in pneumonia we shall very often find corresponding inflammation of the solitary and Peyerian glands of the ileum. Reciprocally, of all the complications of Enteric Fever, pneumonia is the most common. In some stage or degree, I believe, it is very rarely absent. In many cases the inflammation does not proceed beyond active congestion, the post-mortem evidences of which are engorgement with some friability, and the so-called "splenization or carnification."[1] According to the observations of Louis, inflammation of the lungs is more frequent in Enteric Fever than in any other acute disease. He found that splenization, simple or complicated, with partial inflammation of the lung in the first or second degree, existed in twenty out of forty-six cases of Enteric Fever, and in seventeen there was actual inflammation. In only fifteen cases were the lungs healthy, or their alterations slight, little extended, and consisting chiefly of change of colour, due apparently to diffuse or partial congestions. Thirty-eight of his forty-six patients had cough at some period or other of the disease. (Louis, Recher. Fièvre Typhoïde, vol. i. p. 330 et seq.) These observations of Louis are in accordance with those of every other observer. In upwards of thirty cases examined by myself I have found the lungs free from the effects of more or less extensive inflammation only twice. The following case shows the intimate association of the two diseases. It is given by M. Forget as an example of "Follicular Enterite of the inflammatory form:"—

Case 7.—"A strong woman, aged 23, after exposure to hard work in the open air, experienced a sense of painful weariness, headache, nausea, vomiting, thirst, shiverings followed by heat, &c. *Third* day: diarrhœa. *Fourth* day: face flushed; skin hot and dry, pulse frequent, large, resisting; respiration frequent, without cough or pain; tongue white at the centre, red at the edges; abdomen indolent; two liquid stools to-day. *Fifth* day: tongue red, denuded; meteorism; a liquid stool in the night. *Sixth* day: pulse 120, a little nocturnal delirium, dyspnœa, thoracic sibilance. *Seventh* to the *twentieth* day: continued in a typhous condition, with purging, dyspnœa, and more or less delirium. *Twenty-first* day: delirium, groanings during the whole night, deglutition difficult, several liquid stools, pulse frequent, thready; dyspnœa extreme; death.

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[1] M. Louis does not consider "splenization or carnification" as the result of inflammation, but such a condition developed during a general and continued febrile action cannot be regarded as being wholly independent of the inflammatory process.

Necroscopy.—*Head*—notable injection of meninges. *Chest*—old pleuritic adhesions; both lungs engorged—a condition which appeared to have existed for some time—indurated, friable behind and at the bases. *Abdomen*—partial injection of the mucous membrane of the stomach and intestines. Towards the cæcum were met with, at first fine reticulated and swollen Peyer's glands, then rounded ulcerations, which became confluent, confused, fungous in the neighbourhood of the ileo-cæcal valve and upon it: large intestine also presented traces of inflammation and numerous ulcerations, smaller, but more numerous than in the small intestine, and occupying almost its whole length. Mesenteric glands engorged, brownish. Spleen slightly enlarged, friable. Walls of the mouth and pharynx covered with a white pultaceous matter. (Traité de l'Entérite folliculeuse. Obs. lv. p. 414.)

In this case diarrhœa and dyspnœa appear to have commenced simultaneously. The patient died of pneumonia. Take away disease from the one lung, and truly we have, as far as the symptoms and morbid changes are concerned, a typical case of Enteric Fever. Are we, therefore, to attribute the lung disease in this case to a specific *typhoid* poison, the presence of which must be assumed to be proved by the intestinal lesion? Or may we not regard the pneumonia and enteric disease as mere local manifestations of one common inflammatory condition, probably produced by cold? We are inclined to adopt the latter view. In the outbreak of Enteric Fever in the two companies of soldiers under Dr. Grossheim's care, this acute observer could find no other cause for the disease, but "the violence and continuance of the military exercises, and the necessary exposure to great cold after being overheated by violent and laborious corporeal exertions." (Edin. Med. and Surg. Jour. vol. xlviii. p. 187.)

For the association of pneumonia and Enteric Fever, see also cases and 19.

Pleurisy is almost as often present in Enteric Fever as pneumonia. Dr. Murchison observed recent adhesions, or effusion of lymph, in six ut of nineteen cases; Sir W. Jenner, in six out of fifteen; and M. Louis, in two out of forty-six; but he found a greater or less amount of reddish, serous effusion in the pleural cavities in nineteen other ases. (Murchison, p. 560.)

Case 8.—Julia Hatch, aged about 30, died of pleuro-pneumonia on he *forty-fifth* day of the disease. She was admitted into the hospital n the *fourth* day, when the following note was made:—Pulse 120, ongue moist and furred, skin cool and moist, face flushed, respiration ccelerated, slight dulness on percussion, and pleuritic friction sound at the base of the right lung behind; bowels regular. She improved, and took food with a relish until the *twenty-fourth* day, when the pleurisy attacked the left side; the febrile symptoms increased, respiration became hurried and oppressive. A second blister and mustard poultices were applied to the chest, and on the *thirty-sixth* day pulse was 144, feeble; respiration much easier; face less livid; tongue clean; mucous crepitation and friction sound still heard, both before and behind, on both sides. Eats mutton-chops well. After this date she continued to get worse. On the *forty-first* day, pulse 144; respirations 50, laboured; skin hot and very dusky; dulness, crepitation, and friction sound still heard. She continued in the same state till the day of her death. The condition of the skin, tongue, and abdomen was carefully noted from day to day, but, throughout, the digestive function was most regularly performed, the bowels acted naturally every day, and the motions were perfectly healthy. Considering her febrile condition, her appetite for food was unusual. On the *thirty-fifth* day she asked for meat, and enjoyed it. The abdomen was flat and natural, and there was never any appearance of rash.

Autopsy.—Body considerably emaciated; abdomen flat. *Chest*—costal cartilages partially ossified; extensive pleuritic adhesions on both sides, some of which were old, others evidently the result of the last illness; lower lobe of right lung adherent to the diaphragm and side of chest, soft, friable, and slightly crepitant, evidently recovering from recent inflammation. No trace of tubercle in any part of the lungs. Heart healthy, containing soft, yellow clots in the right cavities. *Neck*—fibrinous and serous exudation in the areolar tissue, around the trachea, and between the muscles in front of it. Slight œdema of the mucous membrane above the glottis, and redness of the trachea. Follicular glands at the base of the tongue much enlarged, with violet, swollen, everted margins and gaping orifices. Tonsils a little enlarged. Uvula much swollen. *Abdomen*—intestines undistended and undisturbed; the coils of the small intestine dark purple. The whole of the ileum was intensely inflamed, and every Peyerian gland swollen and prominent. Those in the lower three feet of the bowel, and also the intervening solitary glands, were greatly swollen and ulcerated—whole patches being excavated into ragged ulcers, with rounded, everted, intensely vascular borders, overlying the contiguous mucous membrane; the irregularly excavated centres were deeply stained with bile. One ulcer, two inches from the ileocæcal valve, alone extended to the muscular coat, exposing a smooth surface, half an inch in diameter, of soft, swollen, muscular fibres. The non-ulcerated patches formed elevated fungous-like expansions. Many of the enlarged solitary glands were deeply excavated at the centre. The intervening mucous membrane was excessively vascular. The cæcum was congested, the large intestine healthy, and contained well-formed, solid, bright, yellowish-brown fæces. Mesenteric glands much enlarged, congested, and soft. The spleen, liver, kidneys, supra-renal capsules, and organs of generation were perfectly healthy. The gall bladder was full of healthy, green, viscid bile. Although the patient was subjected every day to close scrutiny, there was no suspicion of intestinal mischief at any time; there was not the faintest external indication of it, but the reverse. I examined the case chiefly with the view of ascertaining how far Peyer's glands are affected in acute disease, and I was surprised to find ravages much more extensive than are seen in ordinary cases of Enteric Fever, and such as would be considered to be eminently typical of this disease.

Laryngitis is a rare complication of Enteric Fever. I have noted it in two cases. In one it occurred during the height of the disease, and yielded to leeching and blistering. In the other it came on during convalescence. Suffocation impended for two days, but the patient escaped by the ejection of fragments of a tough, organized membrane.

Scarlatina and Diphtheria.—A very close relationship appears to exist between these diseases and one variety at least of Enteric Fever. All are, for the most part, autumnal diseases, and they may be observed to increase and decrease together, and all appear to arise spontaneously out of the same conditions. Stöber, Löschner, and Friedleben maintain that scarlatina and Enteric Fever prevail epidemically in an inverse ratio to each other, the one prevailing in proportion as the other declines. (Brit. and For. Med.-Chir. Rev. July 1858, p. 162.) I have known several instances of scarlatina or diphtheria affecting one member of a family and Enteric Fever another, simultaneously. The day before C. B. (case 1) came into the hospital, her brother, aged 14, was admitted with "scarlatina in its most marked form." Sore throat, accompanied by the exudation of white pultaceous matter upon the mucous membrane of the fauces, frequently accompanies the early symptoms of Enteric Fever (*e.g.* cases 4 and 7). Diarrhœa is often a severe complication in scarlatina, and in almost every fatal case of this disease inflammatory swelling of the solitary and agminated and of the mesenteric glands will be found. After most careful microscopical examination of these swollen glands, I have failed to distinguish the slightest difference between them and those of the first period of Enteric Fever. Scarlatina, I have reason to believe, often lapses into Enteric Fever, and such appears to have happened in the case of Julia Hatch (case 8). I have described this case under Pleurisy, but it is probable that its appropriate place would be under Scarlatina. I failed to get information as to her previous history, but the condition of the glands at the root of the tongue, and the neighbouring inflammatory effusions, correspond exactly to the effects of scarlatina. Dr. Murchison noted the co-existence of scarlatina and Enteric Fever in eight cases, and the appearance of scarlet rash without sore throat in five other cases (pp. 518, 473). Other observers have frequently noticed the same facts. Barthez and Rilliet noted the co-existence of diphtheria and Enteric Fever in six cases, Forget in two, Louis in three, and Murchison in one.

The following case of "malignant scarlatina," associated with the anatomical lesions of Enteric Fever, is related by M. Forget :—

Case 9.—A strong man, aged 20, after his usual work, was seized with shivering; during the night, sensation of constriction in the throat, headache, fever, &c. *Third* day: carried to the hospital; hands, forearms, thighs, and chest 'offrent une belle coloration scarlatineuse;' skin burning hot; pulse 140, small; tongue red, and covered, as well as the mouth, with a white, pultaceous coating; throat painful; deglutition very painful; no diarrhœa; chest normal. *Fourth* day: partial stupor; eyes injected; coloration of skin persists; sudamina; pulse 160, thready; pultaceous coating of mouth diminished; back of throat very red, swollen; deglutition almost impossible; epigastrium tender; death.

Necroscopy.—*Head*—meninges injected. *Chest*—lungs engorged throughout, a little friable at the summit; heart filled with white clots. *Abdomen*—gastric and duodenal mucous membrane red, manifestly inflamed; small intestines presented, on approaching the cæcum, numerous Peyer's glands, of which some were simply dotted black, others were reticulated; the majority were red, swollen, firm, elastic, and prominent (gaufrées); an abundant miliary eruption (psorentérie) in a great extent of the small and large intestine, including the rectum.

"Ces caractères anatomiques sont tous ceux de l'entérite folliculeuse très développée, avant la période de gangrène et d'ulcération." (Forget, Obs. xix. p. 144.)

On the next page but one, the same observer gives the history of a case of "scarlatina suive d'entérite folliculeuse."

Such is the association which subsists between scarlatina and Enteric Fever—an association closer and more frequent than is observed between the former and any other acute disease, and one which compels us to acknowledge some closer connexion than mere accidental intercurrence.

Tracing the connexion still further, we observe that the physiognomy, the character of the febrile action and delirium, and the condition of the tongue, are the same in both diseases. In both there is a tendency to epistaxis, cracking of the lips, desquamation of the cuticle, and dropsy (see Anasarca). In the one disease, the solitary and agminated glands of the upper part of the alimentary canal (the tonsils and intervening follicular glands of the tongue), and the neighbouring lymphatic glands, are affected; in the other we find the corresponding parts (the solitary and agminated glands of the ileum, and mesenteric glands) of the lower portion of the alimentary canal diseased. Whether, therefore, we consider these two diseases in reference to their origin, their mode of development, or their physiological anatomy, we still find in either case a resemblance between them. It is only in their subsequent progress that we recognise a clear distinction.

This distinction has reference, *first*, to the nature of the contagious poison—scarlatina tends to spread as scarlatina, and contagious Enteric Fever as Enteric Fever; and *secondly*, to the progress of the two diseases—the one falls upon the cutaneous surface, the other upon the mucous. With regard to the first point, there is nothing in the history of the contagious diseases—at least of the diseases here compared—to dissuade us from the assumption that the contagious animal poisons are developed within the body, and derive their specific characters from the particular actions to which they may happen to be there subjected. Thus, for example, putrescent substances admitted, on the one hand, in a volatile form by the respiratory surface into the *arterial* blood, may be conceived to undergo, during the process of absorption, some special and definite change, whereby a specific poison is formed: and, on the other hand, if the same deleterious agent be taken in a liquid or solid form into the alimentary canal, and thereby admitted into that limited portion of the *venous* system—the portal circulation—we may reasonably assume that it may be peculiarly modified by the agency of the digestive secretions, so as to constitute, upon its admission into the blood, a poison different from that formed in the lungs, but somewhat related to it in its action.

But even assuming that the septic agent be not so diversely modified in the process of absorption, we may still find an explanation of the differences which ultimately distinguish the two diseases if we consider that, in the one case, the liver, a most potent converting agent, intervenes between the portal and systematic circulations, and that by its agency the skin and kidneys—the arterial organs most affected in scarlatina—may be in a great measure protected from a poison introduced by the alimentary canal. If the septic poison be simultaneously admitted into the blood by the lungs and intestinal surface, a mixed disease—scarlatina complicated with Enteric Fever, or the converse—may upon this theory result.

Those who have seen most of these two diseases, and have studied them side by side, will, we feel sure, be most ready to acknowledge how soon their distinctive characters become lost in the intermediate modifications which are observed to occur between them.

The allied affection, croup, is also an occasional associate of Enteric Fever. The following case is taken from M. Louis's work on Typhoid Fever:—

Case 10.—*Croup.*—A powerful man, aged 23, was attacked with slight pain in the throat, preceded by fatigue, lowness of spirits, anorexia, thirst, diarrhœa, and slight epigastric pains. *Third* day: shivering, heat, and sweating; diarrhœa each day very considerable; no sensible increase of the pains in the throat. *Fourth* day: considerable diarrhœa. *Seventh* day: considerable epistaxis; pains in the throat; soft palate red, without swelling; deglutition difficult, and often excited; a sense of prickling and heat in the affected part. *Eighth* day: pain in throat continued; a shining semi-opaque false membrane upon the tonsils, sides of the uvula, which is inflamed, and upon the pharynx; voice a little changed; pain and difficulty of deglutition; four stools and copious sweats during the night; some lenticular rose spots upon the abdomen. *Ninth* day: false membrane more opaque, voice anginose, larynx a little tender, respiration a little accelerated. *Tenth* day: false membrane extending; deglutition causes insupportable anguish. *Eleventh* day: very fœtid breath; croupal voice; deglutition impossible. *Twelfth* day: delirium and death.

Necroscopy.—Cervical glands enlarged to thrice their size and inflamed; false membrane upon the pharynx, the uvula, the soft palate, the epiglottis, and larynx; œsophagus healthy; mucous membrane of stomach and small intestine thickened and softened, and elevated by a kind of white granulations, miliary in the neighbourhood of the duodenum, then proportionately larger as the cæcum was nearer; Peyer's patches more or less red and thickened in the ileum, their thickening being due to swelling of the mucous membrane and subjacent cellular tissue; mesenteric glands large, of an amaranth red, especially near the cæcum, where they were softened; spleen thrice its natural volume.

"As to the symptoms peculiar to Typhoid Fever, if they were little marked, they announced, nevertheless, from their commencement, that the seat of the disease was in the abdomen." (Louis, Obs. xx. p. 187.)

Erysipelas is not infrequently associated with Enteric Fever. Out of 199 cases of Enteric Fever, observed by Louis, Chomel, and Jenner, erysipelas was noticed in twenty. The following is given by Forget as a case of Enteric Fever. We would rather regard it as a case of erysipelas and phlebitis.

Case 11.—A strong man, aged 38, was under treatment in the surgical wards for erysipelas of the left hand, and on a certain day, when the inflammation was in process of resolution, he was seized, without known cause, with shivering, followed by heat, headache, vertigo, nausea, thirst, diarrhœa, and considerable prostration. He was transferred the same evening to the medical wards; expression stupid, sub-icteric tinge, abdomen tympanitic, gurgling, tenderness in right iliac fossa. *Second* day: same state; five liquid stools. *Third* day: pulse 100; six liquid stools. *Fourth* day: same state; diarrhœa; trembling of hands. The patient had been bled on the first day, and to-day one of the punctures is found gaping and exuding a puriform fluid, and the forearm and arm invaded by an inflammatory œdematous swelling very painful on pressure. *Fifth* day: pulse 120; prostration and stupor increasing; several liquid stools; cough; disseminated râles; sub-delirium, and death the same evening.

Necroscopy.—Jaundiced tinge of skin, right arm considerably swollen: pus exudes on pressure from the gaping wound in the vein; vein thickened for length of two inches above the wound. *Chest*—lungs healthy, except a little posterior engorgement. *Abdomen*—alimentary canal healthy to within two feet of the ileo-cæcal valve; Peyer's glands are met with reticulated and swollen; in the intervals a slight psorentery; nearer the valve, and upon it, several patches are in the same state, but redder, more swollen, evidently inflamed, as were the surrounding parts of the intestine; mesentery contained glands swollen and reddish; spleen very large and friable, and no trace of purulent absorption anywhere.

"Voila, certainement, une entérite folliculeuse bien constatée, au cinquième jour." (Forget, Obs. xi. p. 119.) We confess that we cannot see more than the participation of delicate cellular organs—the solitary and agminated glands, the mesenteric glands and the spleen—in a general febrile condition. Had Peyer's patches been found in an ulcerated condition, the erysipelas, which is clearly the primary disease in this case, would probably have been considered to be a secondary complication of latent Enteric Fever.

The following case shows such implication of the solitary and agminated glands in a general inflammatory condition, as is very common in acute disease.

Case 12.—Mary W., aged 40, came under my care for erysipelas of the head and face, August 12, 1865. She had continued pyrexia and muttering delirium. Pulse 116 to 136; the urine was often retained, and the bowels were confined; tongue dry and brown; evacuations sometimes passed involuntarily; urine contained a little albumen; the stools were solid and natural; she died on the *tenth* day after admission. *Head*—brain healthy, but the vessels congested; two ounces of serum in the ventricles. *Chest*—lungs congested and carnified below and behind; healthy in front; heart normal. *Abdomen*—stomach, save a little finely dotted patch of ecchymosis, duodenum, and jejunum, healthy. Lower portion of the ileum and commencement of the large intestine injected. Peyer's glands in the last three feet of the ileum a little swollen and prominent; the intervening mucous membrane strewn with enlarged solitary glands the size of hemp-seeds, giving to the finger, as it passed over the inflamed membrane, a granular sensation. The mucous membrane of the transverse colon, corresponding to one of the longitudinal bands, was highly inflamed. Some Peyer's glands, higher up in the intestine, were stained with sulphide of iron, and were dotted with black. Here and there the contiguous mucous membrane was also stained black. She had taken perchloride of iron. The colon contained solid natural fæces. Spleen weighed five ounces, and both it and the mesenteric glands were natural in size, colour, and consistence. Kidneys and pancreas congested. Liver weighed two pounds fourteen ounces; it was pale, soft, and greasy; bile pale yellow.

Erysipelatous œdema of the glottis has occasionally caused death in Enteric Fever. Several cases are recorded by Jenner and Trousseau.

Case 13.—*Myelitis.*—"A delicate woman, aged 32, was seized at the catamenial period with sharp pains in the loins, the sides, and lower extremities. Menstruation continued the usual time, but the pains persisted and increased. *Fifteenth* day: dorsal decubitus, headache, prostration; face expressive of pain; moans and cries; the least movement is painful; the patient cannot be made to sit for the examination of the spine, which is the seat of sharp pains in its whole extent; the joints are equally painful; the muscles and skin are everywhere extremely sensitive; prickling sensations in the hands and soles of the feet; she cannot stand; skin hot; pulse 100, small and hard; tongue furred; mouth clammy; anorexia; thirst; abdomen, like all the rest of the surface, tender; one stool a day. We diagnose a cerebro-spinal affection. *Seventeenth* day: acute pains; the patient says she cannot feel her limbs, but when they are touched she complains of extreme sensitiveness; spine painful on pressure throughout its whole extent; bowels confined. *Eighteenth* day: general pains; the patient cannot move. *Nineteenth* day: delirium, plaints; acute general pains; stools and urine involuntary; skin hot; sweating; pulse 112, large and supple. *Twentieth* day: same state; diaphoresis, sudamina, vomitings, and numerous stools. *Twenty-second* day: vomiting ceased; numerous involuntary stools; abdominal gurgling; immobility; acute pains provoked by movement; pulse 120. *Twenty-third* day: continued delirium; involuntary stools. *Twenty-fourth* day: the patient is pale, almost pulseless, and bathed in cold sweats; died this day."

Necroscopy twenty-two hours after death.—*Head*—meninges slightly injected; brain of natural appearance and consistency; a little serum in the ventricles; coverings of the cord much injected; a great quantity of serum flowed from the spinal canal. The *spinal marrow* "est ramollie dans une grande étendue, sans changement de couleur; ce phénomène est évidemment cadavérique." *Abdomen*—stomach inflamed in patches. Small intestines present only some vascular ramifications to within two feet from the cæcum; then granulations (*psorentérie*) appear, then reticulated Peyer's patches, others ulcerated very numerous; some appear to be cicatrizing, and some already cicatrized; in the large intestine some isolated follicles appear to be affected; mesenteric glands engorged; spleen and liver present nothing of importance; the bladder contains turbid fœtid urine, and its mucous membrane is strongly injected and dotted. (Obs. xxviii. p. 286.—Forget.)

M. Forget had headed this case "Entérite folliculeuse latente, prise pour une affection cérébro-spinale. Forme, rheumatismale." And after he has detailed the symptoms and post-mortem appearances transcribed above, he asks, "Where is the practitioner who would not have been deceived, as we have been, by appearances so fallacious? How recognise a case of follicular enteritis under such a predominance of sensitive nervous phenomena?" Only by regarding lesions of Peyer's patches so long and so exclusively that no other pathological condition can be conceived possible, we answer.

M. Forget attributes the softening of the cord to post-mortem changes, but the cord is not softer than the circumferential parts of the brain; it is equally well protected from maceration by its vascular coverings, and, from its situation in the axis of the body, it is less liable to post-mortem changes than the brain itself, which, in this case, is described "de consistance et d'aspect naturels." We know of no symptoms, or post-mortem appearances, which could more positively assure us of the existence of inflammatory softening of the cord, than those which the eminent Strasburg professor here places before us.

Phthisis.—In order to illustrate the similarity between the symptoms of tubercular ulceration of the intestines, associated with pulmonary tuberculosis, and those of Enteric Fever, I will here translate two cases from M. Louis's work on Typhoid Fever, and which this eminent author gives as examples, the one of ordinary Enteric Fever, accompanied by delirium, and the other of latent Enteric Fever, but which, with due deference to so great an authority on both phthisis and Typhoid Fever, we feel bound, from our own observations, to regard as cases of tubercular disease. We might have adduced instances in which the lungs were more extensively diseased, from our own practice, but we prefer to place before the reader the description and conclusions of some other author. The reader will form his own opinion on the nature of these two cases, bearing in mind the frequent coexistence of tubercular disease of Peyer's and the solitary glands in pulmonary phthisis, and the difficulty which exists in distinguishing acute tubercular inflammation and ulceration of the glands of the ileum, from the corresponding lesions of ordinary Enteric Fever.

Case 14.—A young woman, aged 17, "d'un embonpoint médiocre," had had cough for four weeks, and in consequence of afflicting intelligence suffered headache, loss of appetite and strength, thirst, increased heat, constipation; the headache was relieved by leeches, the other symptoms continued to the *eighth* day. She took some ipecacuanha, which produced bilious vomiting and purging, with pains in the hypogastrium; cough a little increased. *Ninth* day: sleepy; slight delirium at night; belly everywhere tender on pressure, supple; no tympanites; one stool; pulse 105; intense dry heat; some lenticular rose spots on the back and anterior and lateral parts of the chest; cough moderately frequent; oppression of the chest, mucous râles; in the evening tranquil but profound delirium. *Tenth* day: partly recovered consciousness; tongue red and moist; abdomen tympanitic, a little tender on pressure; heat considerable; startings continue. *Thirteenth* day: pulse weak; mucous sputa, some streaked with blood; a little crepitation at base of right lung behind; continuation of the involuntary movements and meteorism; continued drowsiness during the day, and delirium at night. *Fourteenth* day: meteorism decreased, three or four involuntary stools; crepitant râles heard over the sides of the chest. *Fifteenth* to *nineteenth* day: profound drowsiness and delirium at night; on the evening of the *nineteenth* day respirations much embarrassed, 60. *Twentieth* day: delirium and ineffectual efforts to put the arms out of bed; death.

Necroscopy.—*Head*—brain and meninges apparently normal, only moderately injected. *Chest*—lungs free, filling the cavity of the chest, of a tender rose-colour in front, a little engorged behind for some extent, strewn internally with a great number of grey semi-transparent granulations; bronchi injected, their last divisions covered with a puriform secretion. *Abdomen*—mucous membrane of the small intestine very soft, of an obscure red near the cæcum; Peyer's patches were only visible in the ileum; those nearest the jejunum were pale and obscure, the rest red and successively more developed, larger, and more thickened in proportion as they were nearer the ileo-cæcal valve; those in the last foot of the ileum were ulcerated, their mucous membrane more or less destroyed, and in some the muscular fibres, which were red and thickened, were discovered. The non-ulcerated plates were about a line in thickness. Between Peyer's patches were others much smaller, irregular, and otherwise resembling them, and some yellow, miliary granulations. The last two inches of the mucous membrane were entirely destroyed around nearly the whole of its circumference, and the submucous cellular tissue was more or less red and thickened. Large intestine: mucous membrane thickened and very soft, and presented a considerable number of greyish, lenticular spots, marked with a black point in their centre. All the mesenteric glands were red and livid, and those near the cæcum very large and soft. The mesocolic glands were in the same condition. The spleen was nearly double its volume. (Louis: Obs. xxxiv. p. 25, vol. ii.)

It is strange that Louis, of all other observers, should consider the foregoing to be a case of Typhoid Fever, when the history and anatomical characters are so plainly those of acute tuberculosis. The nature of the other case is still more apparent.

Case 15.—A spare man, aged 25, having short breath from the age of ten years, is taken with the following symptoms:—Disgust of food, thirst, cough, shiverings followed by heat. These symptoms continued, with constipation, until the *twenty-first* day, when he was admitted into the hospital of La Charité. He presented the same symptoms with slight oppression at the epigastrium; constipation still; heat of skin a little exalted, general moisture; pulse large and moderately full; cough infrequent; some mucous expectoration; natural respiratory murmur; moderate weakness. *Twenty-fifth* day: some lenticular rose spots on the abdomen and chest; pulse 86. Up to the *thirty-seventh* day the patient continued stationary, then for the first time he had spontaneous diarrhœa, and grew paler and weaker. *Thirty-eighth* day: a sudden and violent pain in right testicle and corresponding part of hypogastrium, accompanied by a little shivering. The pain came on again in the night, and his slender body was covered with large drops of sweat; he had neither nausea, nor vomiting, nor tympanites; pulse 104. *Thirty-ninth* day: copious diarrhœa; several vomitings of green bile; sweats and pain continued all day. These symptoms continued up to the *forty-fifth* day, when there was great abdominal pain and vomiting. He died this day.

Necroscopy.—Considerable emaciation. *Head*—slight sub-arachnoid effusion. *Chest*—the summit of the left lung presented some cellular adhesions, was a little hard and unequal, and offered for the depth of two inches a considerable number of grey, semi-transparent granulations, in the midst of which a tuberculous excavation was found, the size of a nut, partly empty, and communicating with the bronchi. Below, the pulmonary tissue was in the normal condition. The right lung was in the same condition. *Abdomen*—General peritonitis from perforation of the small intestine about five inches from the cæcum red patches, due to injection of the peritoneum upon the external surface of the small intestine; internally, this part of the bowel presented, at about twenty-four inches from the cæcum, a transverse ulceration about ¾ths of an inch in extent, opposite the mesentery having the attenuated muscular coat for its base, and the edges were not very prominent, and slightly greyish. Six similar ulcerations existed in the last six inches of the ileum, and in the centre of the first of them the perforation, measuring about a fourth of an inch in diameter, was found. The edges of this ulcer were very thin, and partly formed by the peritoneum alone.

The mesenteric glands were a little red, and three or four times their natural volume, and had only half their usual consistence; liver a little pale and soft; spleen twice its natural volume, a little pale and soft.

"The thirst, anorexia, pains in the head, and the shiverings clearly indicate the commencement of the illness in this case. It was only after three weeks that the pains in the belly were experienced Diarrhœa came on as late as the thirty-seventh day. The ulcerations of the intestine being the most profound and without doubt the oldest lesions observed, to these ought to be attributed, in great part at least, the febrile symptoms present at the commencement. It is requisite, however, to remark that the tuberculous affection commenced, according to all appearance, with the principal disease; yet as the ulcerations of the small intestine had the characters of those which occur in the course of the typhoid affection, and as the state of the mesenteric glands[1] could only be referred to that condition, this disease has evidently had the greatest share in producing the symptoms and lesions observed, and we can only place this case in the chapter upon the latent typhoid affection." (Louis, Obs. xliii. p. 232, et seq.)

Endemic Intermittent and Remittent Fevers.—One of the most general facts observed in reference to Enteric Fever, is the frequent occurrence of intermittence in the pyrexial condition. The commissioners appointed to investigate the French epidemics of Enteric Fever, "call attention to the fact that a more or less pernicious intermittent, or at least remittent character, was manifested under a great variety of circumstances." (De Claubry, Mém. de l'Acad. de Méd. tome xiv. p. 71.) "A great number of cases of Typhoid Fever presented, either at the commencement of the disease, transient symptoms of simple intermittent fever, or during its further progress, intermittent or at least remittent phenomena, which rendered the employment of quinine necessary." (Ibid. p. 11.) M. Trousseau (Clinique Médicale) records cases to show that "Enteric Fever may simulate at first intermittent fever, and reciprocally, an intermittent fever may assume at the commencement the characters of Typhoid Fever." (P. 247, 2d Edition.) "It is especially in countries where marsh intermittent fevers are endemic, and with individuals who have recently left their own country, that we see Enteric Fever assume at its commencement an intermittent type." (P. 250.)

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[1] Louis appears to regard a vascular, swollen, and softened condition of the mesenteric glands as being inconsistent with the existence of tubercular disease. We have frequently seen the mesenteric glands purple, soft, and swollen in cases of advanced phthisis in which both lungs and intestinal glands have been affected. (See Diagnosis.)

Intermittent fever is, in the present day, nearly extinct in England. Its last strongholds are to be found in the north part of Kent. There, about the Isle of Sheppey, and on the marshy banks of the Swale, it still lingers. At Milton, for example, ague is still common in the autumn. "The drinking water is obtained from wells, and the general sanitary condition, as regards drainage and the non-removal of nuisances, is unsatisfactory, and there has been a considerable amount of Typhoid Fever at times, and scarlatina in a severe form was prevalent at the time of the inspection." (Rep. by Dr. G. Whitley, as to quantity of ague now prevailing in England. Sixth Rep. Privy Council, 1863, p. 432.)

At Holbeach and Long Sutton, Ague and Enteric Fever were both prevalent. The drainage of these places is bad, and the water supply bad, being from pits (p. 441). "Very nearly all the medical men who had had opportunities of forming an opinion concerning the co-existence of ague and typhoid fever in the same districts, were of opinion that the local conditions which produce the former are favourable to the development of the latter. Thus, Mr. Keddell, with forty years' experience in Sheppey, believed that when ague, from certain conditions of surface, is rife in summer, bilious, remittent, and typhoid fevers prevail in autumn." (Ibid, p. 452.)

My friend Mr. Charles Mayo informs me, from extensive personal observation, that the "camp fever" of the army of the Potomac was generally recognised as a "typho-malarious fever," in which the symptoms of typhoid fever, diarrhœa, rose rash, &c. were associated with those of intermittent fever. The typhoid symptoms occasionally predominated, and post-mortem examination revealed lesions of Peyer's glands.

It is evident from the foregoing observation that an investigation into the nature of Enteric Fever would be very incomplete without a brief consideration of the symptoms and morbid anatomy of the severer forms of intermittent fever. The Walcheren fever offers itself as a standard of comparison. Dr. Davis[1] has given a very clear and minute description of this disease. "The Walcheren fever," he says, "assumed the quotidian, tertian, double-tertian, and even remitting type. It did not uniformly declare itself with the same type, being one while continued, then remittent or intermittent, and changing its type again from these to the continued character. I believe the

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[1] View of the Fever of Walcheren and its Consequences, by J. B. Davis, M.D. 8vo. Lond. 1810.

Walcheren fever in many instances would have ceased but for the derangement it had occasioned in the abdominal viscera, becoming in some measure a secondary disease." (P. 12, et seq.) The *premonitory symptoms* were weakness, nausea, headache, universal languor, dejection of spirits, always combined with a vitiated state, suppression, or diminution of the intestinal and biliary secretions.

After the paroxysms, headache, confused intellect for two or three days, ending in coma and torpor. At other times continued pyrexia, whiteness of the tongue, distension and uneasiness of the epigastric region, and anorexia. Then the bowels became painful, and there were diarrhœa, discharge of mucus, or much blood intermingled with fæces, &c. &c. These symptoms would be obscured by the paroxysm, to reappear after it was over (p. 18). "All the patients with the quartan type under my care were very prone to diarrhœa and dysentery, thirst, pyrexia, emaciation, daily exacerbations of hectic, local pains, and general irritation, constituting an unmanageable disease which wore away the patient's strength, and utterly exhausted him" (p. 17). Delirium was seldom formidable; epistaxis frequently occurred. Hectic was almost uniformly the character of the pyrexia. Grey, clay-coloured watery stools, and rapid marasmus, were common in cases tending to a fatal termination.

Such were the symptoms of "the continued fever or long-continued paroxysm," when the order of the periods became so completely overturned that it was difficult to bring the fever to its proper type again (p. 21).

If we now turn to the morbid anatomy of the disease, we shall find positive evidence of lesion of the solitary and agminate glands of the small intestines, in at least six of the cases recorded by Dr. Davis. Usually both the small and large intestine were involved in the disease, and its ravages greatest in the latter. Thus, in case 29, we have a description of the post-mortem appearances of dysentery implicating the small intestine:—Colon ulcerated throughout its whole extent. Rectum much ulcerated and had sloughed near its termination. Jejunum and ileum "interspersed with black spots internally; the intervening spaces red, and raised up into little protuberances, resembling granulations of flesh, not unlike a cock's comb." Liver large, black, and soft. Spleen soft, of enormous size. Mesenteric glands enlarged (p. 173).

In case 32 the large and small intestines appear to have been pretty equally affected. "The small intestines were of a deep purple colour, and interspersed on their inner side with tubercles and small ulcers, resembling chancres. The colon and rectum had numerous tubercles and ulcerations." Spleen weighed four pounds, and was uniformly soft throughout. Mesenteric glands enlarged (p. 175).

In case 34 the lesions were more apparent in the small than in the large intestine. The convolutions of the small intestines were united together by condensed coagulable lymph. They were of a reddish colour, and interspersed with small red eminences on their inner surface. The coats of the colon were thickened. Liver large and black. Spleen firm and dark, weighed four pounds.

The particular characters of the intestinal lesions are thus clearly described by Dr. Davis. The ileum and jejunum were frequently interspersed with "tubercles inflamed and ulcerated in different parts. Here and there small eminences of the size of a pin's head, or round bodies with an ulcer at the point, or little ragged ulcers, excavated in the middle, resembling chancres, or one large, or a succession of small ulcers spreading wide upon, and deep into, the coats of the intestines. Colour of these tubercles various, consistence firm. While their points were yellow their edges were hard, and their bases almost black, like a lump of decayed flesh. They did not come fairly to suppuration, but appeared gradually to crumble away and degenerate into a scabrous ulcer. These bodies had their origin beneath the villous coat of the intestine." (P. 191.)

From the above description we may infer that the morbid condition of the solid and agminate glands of the intestine in these fatal cases of intermittent fever is identical with that which is assumed to be characteristic of Enteric Fever. We have already seen that both forms of fever are developed amidst the same conditions, and we therefore unhesitatingly conclude that Enteric Fever is often a part of intermittent fever, and the converse.

Dysentery.—After the foregoing observations it may appear superfluous to call special attention to the relation between Dysentery and Enteric Fever. But the connexion between these two diseases is too important to receive only a cursory notice. Even in reference to Enteric Fever alone, it is important to observe that the ulceration sometimes spreads to the large intestine, when the lesions of the small intestine are in process of reparation. The following isolated case recorded by Forget, may be briefly mentioned to show how the enteric disease may be prolonged by subsequent lesion of the large intestine.

Case 16.—A patient was laid up with the usual symptoms of Enteric Fever for a month, then, after a few days' intermission, profuse dysenteric diarrhœa, tenesmus, and colic pains set in, and after continuing for about twenty days killed the patient. In the last two feet of the ileum, "numerous white shining spots, of variable extent, smaller than the ulcerated Peyer's patches, and evidently cicatrices, were found. The great intestine was profoundly altered in all its extent from the ileo-cæcal valve to the anus; it was brown, black, hypertrophied, vegetant, and softened, presenting ulcerations of various depth." (Forget, Obs. xlii. p. 351.)

Rokitansky describes "the typhous process in the mucous membrane of the small intestine," as distinct from "the dysenteric process" observed in the large intestine. But this distinction is purely artificial. In the following well-marked case of Enteric Fever which lately died under my care, the large intestine was the more extensively ulcerated, and the ulcers in both small and large intestine were indistinguishable from the so-called "dysenteric ulcers."

Case 17.—Catherine M., aged 23, residing at Stanmore, near London, was taken ill this autumn with headache, much shivering, pain in the back, and diarrhœa. Fever and diarrhœa continued; rose spots appeared on the abdomen from the *tenth* to the *twenty-second* day. Diarrhœa persisted, and there was much hectic. On the *twenty-fourth* day there was marked abdominal tenderness. The diarrhœa continued unchecked, and she died on the *twenty-seventh* day.

Necroscopy.—Chest—lungs congested, friable at apices, weighed thirty-three ounces. *Abdomen*—liver enlarged, weighed three pounds five ounces, soft, greasy, and pale. Gall-bladder full of pale, thin, ochre-coloured bile. Stomach, duodenum, jejunum and upper portion of ileum healthy; last two feet of ileum presented sixteen ulcerations of Peyer's glands, varying from minute vascular abrasions to three-eighths of an inch in diameter. Four of these pale depressed ulcers were situated immediately above the ileo-cæcal valve, and were evidently in process of contraction and cicatrization. In the large intestine there were twenty-seven ulcers, twenty of which were in the cæcum; several were situated immediately beneath the ileo-cæcal valve, and one of these was as large as a shilling, and deeply excavated the muscular fibres; another, the size of a sixpence, was placed at the bottom of the cæcal pouch, and it lay upon the peritoneum, which presented externally a corresponding patch of opacity with vascular ramifications. Seven other ulcers occurred at intervals in the ascending and transverse colon, the last one occurring at a distance of two feet from the ileo-cæcal valve. All these ulcers were pale, with ashy or smooth bases lying upon the muscular fibres; their edges were not elevated, and often perpendicular. The solitary glands of the large intestine were enlarged, the central parts of many were eroded and in a state of incipient ulceration. The mesenteric and mesocolic glands were purple, much enlarged, and soft. The spleen weighed six and a half ounces, and was of normal colour and consistence. Kidneys, pancreas, and the other organs healthy.

The day after this young woman died, a patient, in the same ward, under the care of my colleague, Dr. Murchison, also died; and as the case illustrates very well how extensively the large intestine may be ulcerated in Enteric Fever, I have availed myself of Dr. Murchison's kindness in allowing me to make my observations of the case, and briefly detail them here.

Case 18.—Eliza H., aged 26, was admitted on the *tenth* day of her illness with fully developed Enteric Fever. She was taken ill with headache, heats and chills, and diarrhœa, and these symptoms continued to the time of her admission. Rose spots appeared on the abdomen from the *tenth* to the *eighteenth* day. The bowels continued very loose, and the stools were of a light yellow colour. Medicines failed to restrain the diarrhœa, the abdomen became distended and tender, and the patient died exhausted on the *twenty-seventh* day of her illness.

Necroscopy.—Chest—lungs healthy, with only a little hypostatic congestion. Heart contained firm fibrinous clots in all its cavities. *Abdomen*—stomach, duodenum, jejunum, and upper portion of ileum perfectly healthy. In the last two feet of the ileum there were a dozen pale non-elevated ulcerations of Peyer's glands; six of them were in the immediate neighbourhood of the ileo-cæcal valve, and the largest did not exceed three-eighths of an inch in diameter. All were evidently in process of healing. The large intestine was in a state of ragged ulceration from the under surface of the ileo-cæcal valve to within an inch of the rectum. In the transverse and descending colon there were two rows of ulcers, each about a foot long; these ulcers were very deeply excavated, and for the most part confluent, or only separated by narrow bands of hypertrophied mucous membrane. Each ulcer, or confluent patch, was about an inch wide. The edges were two or three lines thick, irregular and very vascular, and often black; the surface of the ulcer was chiefly formed of ashy sloughs of areolar tissue, or disintegrated muscular fibres. In the interval between these rows of ragged ulcers, were a great many circular ulcers, and swollen solitary glands advancing to this condition. Nearer the cæcum and rectum the ulcers were fewer and more discrete. The mesocolic glands were greatly enlarged, purple, and soft. The spleen weighed eight and a half ounces, and was pulpy. The liver was very soft and greasy; it weighed forty-four and a half ounces. The gall-bladder contained half an ounce of pale yellow watery bile, which did not affect turmeric paper, but changed blue litmus to red. The other organs were quite healthy.

In the following case of Enteric Fever and pneumonia, the intestinal lesion was almost entirely confined to the colon:—

Case 19.—Phœbe Poole, aged 14, was admitted into the London Fever Hospital on the 8th of September, 1865, on the *fourteenth* day of her illness. She had had cough, quick breathing, and diarrhœa, accompanied by high fever. At this date the pulse was 150, tongue dry, brown, and cracked, skin pungently hot, respirations 48, cough, dulness with crepitation and bronchophony over the lower lobe of the right lung behind; there was retention of urine, the abdomen was tympanitic and tender, there were two rose spots upon its surface, and the bowels were very loose. The pulmonary and enteric inflammation progressed, tubular breathing was heard over almost the whole of the right side of the chest, and the bowels continued very loose. A few fresh rose spots appeared up to the *nineteenth* day. On the *twenty-ninth* day the respirations were 60, short and snatchy, the pulse 160, and the diarrhœa profuse. On the *thirty-fifth* day she died.

Autopsy.—Chest—right lung completely solid and firm, grey and gangrenous. Left lung a little engorged. Heart healthy. *Abdomen*—the last Peyer's patch near the ileo-cæcal valve had two minute ulcerations, but the rest of the gland, and all the other agminated and solitary glands, were perfectly healthy. The colic side of the cæcum, and the first five inches of the ascending colon, were in a state of ragged ulceration—long, clean, transverse ulcers, laying bare and dissecting the muscular fibres, were repeatedly confluent in this part of the bowel, and were interspersed with islands of soft, greatly-swollen, mucous membrane. Lower down, were ulcerated solitary glands, and there were six more in the sigmoid flexure. The solitary glands and mucous membrane of the rest of the large intestine were generally healthy. The mesenteric glands corresponding to the small intestine were quite healthy; the mesocolic glands in the neighbourhood of the cæcum were purple, soft, and much enlarged. The rest of the viscera appeared healthy. The spleen was of natural size.

Such cases as the foregoing afford typical examples of acute dysenteric ulceration, and we may question whether the distinction between Dysentery and Enteric Fever is not somewhat artificial.

Cholera.—"In the delta of the Ganges, the Nile, and the Mississippi the three forms of disease called cholera, plague, and yellow fever, are constantly seen preceding, accompanying, and following intermittent fever, and constitute there the reigning endemic diseases; and one is forced to recognise a very great analogy, not to say an identity, of origin between the marsh fever and the three great scourges above mentioned." (Traité des Fièvres intermittentes, rémittentes, et continues, par J. C. M. Boudin, p. 161.) A protracted attack of cholera bears a close resemblance to Enteric Fever; the intestinal lesions of the two diseases, moreover, are indistinguishable from each other. "The most frequent of all the abnormal conditions of the mucous membrane of the intestines was prominence of the intestinal glands, both aggregate and solitary, but especially the latter. This condition, the *psorentérie* of some French writers, was found in about two-thirds of the eighty-nine fatal cases examined." (W. T. Gairdner, M.D. Month. Journ. Med. Science, 1849.) M. Pirogoff examined 500 fatal cases of cholera. He observed in the earlier periods of the disease, "thickening and swelling of the mucous membrane most often accompanied by swelling of Peyer's and the solitary glands, as well as swelling of the mesenteric glands. In the typhoid period, ulceration of these glands." (Anatomie Pathologique du Cholera Morbus. Folio, St. Petersburg, 1849.)

Scurvy is sometimes accompanied by ulceration of the solitary and agminate glands, with all the symptoms of Enteric Fever. An outbreak of scurvy occurred in the Milbank Penitentiary, in London, in the years 1822–23. An account of it was published by P. M. Latham, M.D. (8vo. Lond. 1825.) "In addition to the ordinary symptoms of scurvy—purpura hæmorrhagica, spongy and bleeding gums, &c.—there was every degree and species of flux ever seen or described. There were cases which corresponded with the descriptions of the Indian cholera, and there were some which corresponded with the common autumnal cholera of this country, except that they were accompanied by intractable diarrhœa. There was every kind and degree of dysentery." (P. 32, 33.) In some cases the abdomen was soft and natural; in others tympanitic. Post-mortem examinations revealed lesions of the intestines, which, from the descriptions at pp. 46–49, are clearly to be attributed to swelling and ulceration, even to perforation, of the solitary and agminated glands.

VARIETIES.—After the foregoing review of the associated pathology of Enteric Fever, can we adopt the dogma of Chomel?—"Quand nous trouverons dans les auteurs, soit anciens, soit modernes, des observations de maladies aigues à la suite desquelles on aura rencontre des ulcères à la fin de l'intestine grêle, nous aurons le droit de les considérer comme des cas d'affection typhoïde." (Fièvre Typhoïde, p. 113.) Or, going to the other extreme, shall we deny the existence of Enteric Fever as a specific disease, and regard the intestinal lesions merely as the result of an accidental but severe local complication which may arise in any general febrile condition of the body? If we accept the first proposition, we must include Tuberculosis under Enteric Fever. If we adopt the second, we may, with almost equal reason, deny the existence of Scarlatina as a distinct disease. We can only avoid the dilemma by admitting that the enteric disease, and all its attendant phenomena, may occasionally become a part of some other more general inflammatory condition, and then, I think, it can hardly be denied that in other cases the disease is due to some poison or poison introduced from without, in the elimination of which, the digestive organs especially are deranged. In order to include all the phenomena of Enteric Fever, I find it necessary to divide it into these three varieties: (1) Simple Inflammatory Enteric Fever; (2) Contagious Enteric Fever; (3) Paludal Enteric Fever.

It may seem paradoxical and unphilosophical to include under one kind contagious and non-contagious diseases; but since the question of contagion is still an open one with many, and neither symptoms nor anatomical lesions mark a distinction, we must be content thus to classify the disease for the present.

1. *Simple Inflammatory Enteric Fever.*—This variety is non-contagious, due to no specific cause, and may arise in any inflammatory condition of the body, such as accompanies pneumonia, erysipelas, pyæmia, &c. The common enteritis, which constitutes autumnal diarrhœa, if protracted, often lapses into this variety of Enteric Fever. Cases 6, 7, 11, 19, &c. furnish examples of this variety. The recognition of the intercurrence of enteric inflammation, with ulceration of the glands of the ileum, in acute disease generally, is of very great importance; for of all the organs of the body, these delicate glands, from their situation within the thin and vascular intestinal wall, are less capable than similarly constituted parts elsewhere situated, of enduring prolonged inflammation, without risk of fatal accidents; and at any time the intestinal lesion may become much the gravest part of the more general disorder. The frequency with which the intestinal glands become implicated in acute disease is probably due to their exposed situation, their delicate corpuscular structure, their great vascularity, and the arrangement of their blood-vessels.

A well-developed healthy lad, aged 15, fell from a horse; the skull was fractured, and the corresponding surface of the brain lacerated; febrile action followed, and he died on the third day. Before the body was cold I examined the small intestine. In the last nine inches of the ileum I found the solitary glands swollen, and of a delicate greyish-pink colour, and semi-transparent appearance, forming rounded elevations of the mucous membrane, the size of hemp-seeds (psorentery). The mesenteric glands were a little increased in vascularity and size. There was no trace of disease in any part of the body. Here we recognise a condition of the solitary glands, which, under the continuance of the general febrile action, *might* have passed into the worst form of "typhoid ulceration."

"But surely," it will be said, "we can distinguish the true typhoid ulceration from any other at a glance; moreover, the typhoid ulcer is characterised by the deposit of a distinct morbid material—a specific *typhous cell*." Having already shown that the inflammatory swelling is due, not to the deposit of a specific morbid matter within the glands, but to the rapid growth of their normal corpuscular constituents under the influence of undue vascular excitement, we fail to recognise any character by which one form of inflammatory action in Peyer's glands can be distinguished from any other. Ultimately, we shall have little or no difficulty in distinguishing a tubercular ulcer from any other; but between the lesions assumed to be characteristic of Enteric Fever and those arising from ordinary inflammation, which, of course, may affect the intestinal glands in common with every other part and organ of the body, there is, I conceive, no distinction. As a result of common inflammatory action in the glands, we may find Peyer's glands swollen into cock's-comb or fungus-like elevations, and excavated into ragged ulcers, with red everted edges, or occupied by sloughy cores. (See case 8.) In a well-marked case of Enteric Fever, in which the diarrhœa, rose spots, and abdominal pain and swelling call attention to the abdominal lesion, we may, on the other hand, find, as in a case which I examined two days ago, a dozen angry-looking ulcers in the last foot of the ileum, varying in diameter from two lines to three-fourths of an inch, and exposing the red-streaked muscular fibres, each ulcer sharply cut, and the irregular margins *not* raised above the general level of the intervening dark red mucous membrane. Higher up were seen glands level with the inflamed mucous membrane, and presenting sloughy erosions, like an aphthous ulcer of the mouth.

2. *Contagious Enteric Fever.*—Of this variety I can say but little. I am not *sure* that I have seen it; but that it exists, appears to be an indisputable fact. Eight of the sixty-eight patients, referred to below, came from houses in which other residents were affected with the fever. The disease may have been propagated by contagion in some of these cases, but in two instances, in each of which three members of the same family were affected, I found from personal observation, that an endemic cause existed in impure drinking water.

See also cases 4 and 5, and the observations upon "Contagion." The associations of this variety appear to be, as I have already pointed out, with scarlatina and the allied affections, diphtheria and croup. Exudations upon the faucial and laryngeal mucous membrane appear to be frequent in this variety, and the course of the disease more rapid than in the third variety.

3. *Paludal Enteric Fever.*—This we believe to be the common form of the disease. It arises from putrescent animal and vegetable substances. It is non-contagious, and its course is usually slow. Case 2 may be taken to illustrate this variety of the disease.

In thus unreservedly recognising the connexion which, I cannot doubt, exists between Intermittent and Enteric Fever, it may appear to some that I transgress the facts which have been adduced to illustrate this view; but I feel sure that justice has not been done to the numerous observations which abound in medical literature, and which, if collected, would together form irresistible evidence of the direct connexion between these two diseases. In the low-lying districts on the banks of the Thames, within and about the metropolis where ague was formerly so rife, Enteric Fever prevails continuously, becoming very abundant in the autumn, while the higher situations are comparatively free from it. Of sixty-eight cases of well-developed Enteric Fever which have come under my care during the present autumn (1865), fourteen came from the districts of Stanmore, Chelsea, Lambeth, Southwark, Stepney, Hackney, Bethnal Green; thirteen from the lowest part of the parish of St. Luke alone, where cesspools and pumps are still in use, and where drainage works are now in progress; twenty-three from the filthiest and most crowded parts of the parishes of St. Clement Danes, Holborn, and St. Giles; and only five from the more elevated localities of Soho and Marylebone, Islington, Holloway, &c. The remainder resided in Maidstone, Croydon, Mitcham, Edmonton, and various other country districts near London.

We find Enteric Fever remarkably prevalent in the spreading outskirts of the suburbs, where new houses and streets are constantly springing up beyond the limits of the drainage works.

We acknowledge as modifications of the same disease, that intermittent form to which, when London had its cesspools and pumps, and retained all its filth within its undrained area, James I. fell a victim, and that continued modification which still lingers in a subdued form in the same locality, and to which a good Prince has succumbed in our own generation.

Distribution.—Enteric Fever prevails in every inhabited part of the world. No situation is secure from it. In the report of the epidemics which have occurred in France from 1841 to 1846, De Claubry (Mém. de l'Académie de Méd. tome xiv.) observes: "The situation of the twenty-eight departments, which have been the frequent theatre of destructive epidemics of typhoid fever, was such, that it appeared impossible to conclude that it had any influence whatever in the production of these epidemics." (P. 4.) "If one finds typhoid fever on the one hand in villages situated in deep valleys, in narrow gorges; in lowlands, where the watercourses frequently overflow, making the submerged soil, upon which the miserable dwellings are built, extremely damp: one sees it, on the other hand, in villages situated, one upon the most elevated points of a high chain of mountains, and constantly exposed to every wind, and having no unhealthy condition in its neighbourhood; another situated 600 feet above a little flowing stream, commanding an extensive view of perfectly cultivated fields; a third, in a very salubrious position, upon an undulating soil, where the flow of water is perfect." (P. 8.)

Enteric Fever, moreover, attacks every class of society indifferently. On one hand, we find it associated with the most abject poverty, damp, filth, overcrowding, and defective ventilation; and, on the other, we witness the disease making havoc amongst the wealthy residents of spacious, dry, well-built houses, isolated, or united to form wide open streets, or elevated terraces.

CAUSES: (*a*) *Predisposing.*—Of the causes which predispose to Enteric Fever, *youth* is usually considered to be one; but young people are not more liable to this than they are to other inflammatory diseases. Dr. Murchison states (page 409), that slightly more than half of the cases of Enteric Fever admitted during ten years into the London Fever Hospital were between fifteen and twenty-five years of age; one-fifth were under fifteen; less than one-seventh above thirty; and only one-sixty-eighth exceeded fifty. Similar statistical results may probably be found in many other acute diseases.

Seasons have a marked influence on the increase and diminution of Enteric Fever. "Out of 106 times in which an exact indication of the epoch when the epidemics of typhoid fever commenced, the reports of the years 1841 to 1846 give the following results:—First yearly quarter, twenty epidemics; second quarter, twenty-one; third quarter, thirty-nine; fourth, thirty-six: or, summer (April to September), sixty; winter (October to March), fifty-six. Seventy epidemics commenced in the four months of August, September, October, and November; while only forty-six commenced in the other eight months of the year, from December to July." (De Claubry, op. cit. p. 8.)

"In New England Enteric Fever is not infrequently called the autumnal or fall fever." (Bartlett on Fevers, p. 101.)

On examining the accompanying Table (*vide* Table) of the cases which have occurred at the London Fever Hospital during the last 18 years, the following facts appear:—*First*, that the greater number of cases occur during the autumn and winter months, and the average of 17 complete years[1] shows that more than twice as many cases, or a proportion of 2·1 to 1, occur during these periods, as compared with those happening during the other six months of the year.

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[1] From 1848 to 1864 inclusive.

TABLE *showing the number of cases of* ENTERIC FEVER *treated in the* LONDON FEVER HOSPITAL, *and certain attendant atmospheric conditions, during the last eighteen years. The analytical observations have no reference to* 1865, *which year was incomplete when they were made.*

The *first column* gives the number of cases admitted during every month, season, and year. It is partly derived from p. 417 of Dr. Murchison's work, and partly from the London Fever Hospital Reports. The *second column* gives the mean temperature of the air during each month, season, and year. The *third column* shows in like manner the mean degree of moisture contained in the air. The numbers have reference to 100, which is taken to represent the greatest quantity of aqueous vapour which the air can at any temperature retain. The *fourth column* gives the total rainfall in each month, season, and year. The second, third, and fourth columns are from the Registrar-General's Returns, and are due to Mr. James Glaisher, F.R.S.

	1848.				1849.				1850.				1851.				1852.				1853.				1854.				1855.				1856.			
	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.
January .	9	34·6°	83	1·2	9	40·1°	88	1·6	6	33·7°	89	1·2	13	42·9°	85	2·7	10	42·0°	84	3·6	17	42·4°	80	2·0	13	39·0°	91	1·7	16	34·9°	91	1·0	12	39·4°	91	2·6
February .	9	43·4	86	2·6	7	43·2	86	2·2	5	44·7	83	1·3	8	40·1	87	1·2	12	40·8	87	0·9	7	33·3	80	0·9	13	39·5	84	1·0	9	29·4	91	1·4	10	42·0	87	1·1
March . .	7	43·8	83	3·1	5	42·5	80	0·5	5	39·9	77	0·3	12	42·6	84	4·1	6	41·3	81	0·2	14	38·5	78	1·5	7	43·8	79	0·4	5	37·9	86	1·5	8	38·7	82	1·1
April . .	4	Mean temp. & hum. of suc. qr. & totl. rain.			3	43·2	86	2·2	7	48·5	79	2·3	8	44·7	82	2·3	7	45·9	75	0·5	5	45·2	77	3·1	6	48·4	77	0·6	8	45·8	78	0·1	8	46·8	74	2·3
May . . .	4	54·3	81	7·3	4	54·0	70	3·9	14	51·3	76	2·4	16	50·9	76	0·8	9	51·5	77	1·9	6	52·0	74	1·6	8	50·9	85	3·3	10	48·8	80	1·8	7	49·5	77	3·5
June. . .	13	...	...	...	12	57·9	71	0·2	11	60·8	70	0·9	24	58·9	73	1·3	9	56·1	78	4·6	17	58·2	70	2·8	10	55·7	82	1·0	7	56·9	74	0·7	7	58·5	79	1·6
July . . .	16	...	...	...	16	62·1	71	2·9	15	62·2	88	2·9	29	60·1	77	4·2	4	66·6	73	2·3	11	60·3	76	6·0	15	60·3	78	1·7	25	62·1	80	5·0	7	61·1	79	0·9
August . .	17	58·6	78	8·7	16	62·9	77	0·45	13	60·2	80	1·9	18	62·3	76	2·6	22	62·1	72	4·5	33	60·0	77	2·2	20	60·9	77	2·9	40	62·1	76	1·1	28	63·6	78	2·4
September.	26	...	...	...	19	58·8	77	3·3	13	56·4	75	1·3	27	56·9	76	0·5	19	56·8	77	3·9	33	55·3	84	2·4	49	58·1	77	0·7	26	57·1	80	1·1	14	55·2	75	2·8
October .	17	...	...	...	25	51·1	81	2·7	17	47·0	82	1·4	24	52·6	81	1·8	12	47·9	80	3·8	29	50·9	90	4·3	51	49·4	84	2·6	25	51·2	89	4·9	15	51·7	87	1·6
November .	19	45·9	88	7·3	16	44·1	86	1·5	17	46·5	85	2·5	30	37·9	82	0·6	12	48·9	82	6·0	26	42·1	93	1·5	20	40·5	91	1·4	22	41·3	92	1·3	28	40·7	88	1·0
December .	11	...	...	...	6	39·1	90	2·4	14	40·6	92	1·3	25	40·5	69	0·6	18	47·6	79	2·2	13	34·0	91	0·7	16	41·3	87	1·4	24	35·6	79	1·2	5	40·2	90	1·3
(*a*) Spring .	20	...	...	...	15	42·9	84	4·9	17	44·3	79	3·9	28	42·5	84	7·6	25	42·7	81	1·6	26	39·0	78	5·5	26	43·9	80	2·0	22	37·7	85	3·0	26	42·5	81	4·5
(*b*) Summer	33	...	...	...	32	58·0	70	7·0	40	58·1	78	6·2	69	56·6	75	6·3	22	58·0	76	8·8	34	57·0	73	10·4	33	55·6	81	6·0	42	55·9	78	7·5	21	56·3	78	6·0
c) Autumn	60	...	...	...	60	57·6	78	6·4	43	54·5	79	4·6	69	57·2	77	4·9	53	55·6	76	12·2	95	55·4	84	8·9	120	56·1	79	6·2	91	56·8	81	7·1	57	56·8	80	6·8
(*d*) Winter .	39	...	...	...	31	41·1	88	5·5	37	40·2	88	5·0	68	40·4	79	3·9	40	46·2	82	11·8	56	39·5	88	4·2	49	40·2	89	4·5	62	33·9	87	3·5	45	40·1	89	4·9
Total .	152	...	...	23·3	138	49·9	80	23·8	137	40·2	81	19·7	234	49·2	79	22·7	140	50·6	79	34·4	211	47·7	81	29·0	228	48·9	82	18·7	217	46·0	82	21·1	149	48·9	82	22·2

TABLE *showing the number of cases of* ENTERIC FEVER *treated in the* LONDON FEVER HOSPITAL, *continued* :—

	1857.				1858.				1859.				1860.				1861.			
	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.
January .	8	° 36·6	92	2·6	32	° 37·5	86	0·7	13	° 40·4	88	0·8	14	° 39·7	88	1·8	8	° 33·9	85	0·5
February .	5	39·2	87	0·2	7	34·6	84	1·7	10	43·1	81	0·9	6	35·7	80	1·1	8	42·1	91	1·8
March . .	8	41·8	84	0·8	13	41·4	78	0·9	12	46·4	79	1·3	11	41·1	79	1·9	6	43·8	90	2·2
Apri . .	4	45·7	82	1·4	5	46·2	76	2·4	5	46·6	78	2·2	7	42·9	79	1·0	3	44·3	85	0·8
May . . .	1	54·0	74	0·6	9	51·7	75	1·8	5	53·1	77	2·4	4	53·8	75	3·9	1	51·9	74	1·8
June. . .	9	61·8	72	2·7	7	64·9	67	1·2	8	61·4	77	1·4	2	54·8	82	5·8	2	59·1	81	1·9
July . . .	19	64·5	71	1·1	13	60·6	72	2·9	12	68·1	70	3·3	7	57·6	83	2·8	10	60·9	78	2·2
August . .	26	65·8	73	2·6	29	62·0	70	1·6	20	63·5	72	1·1	10	57·7	83	3·7	18	63·2	76	0·6
September.	34	59·7	86	3·4	22	60·3	78	0·9	20	56·7	75	3·8	9	53·4	88	3·1	31	57·1	78	1·5
October .	38	52·9	92	4·2	20	50·8	85	1·2	33	50·9	89	3·6	6	50·6	89	1·6	30	54·9	87	0·9
November .	33	45·9	94	1·3	15	39·6	86	0·4	27	42·1	87	2·9	6	40·8	93	2·5	31	40·8	87	5·2
December .	29	45·0	90	0·5	8	41·0	89	1·5	11	36·8	88	2·2	13	36·3	92	2·8	13	41·0	87	1·3
(*a*) Spring .	17	42·2	84	2·4	25	40·7	70	5·0	27	45·9	79	4·4	24	39·9	79	4·0	17	43·4	88	4·8
(*b*) Summer	29	60·1	72	4·4	29	59·1	71	5·9	25	60·9	74	7·1	13	55·4	80	12·5	13	57·3	77	5·9
(*c*) Autumn	98	59·4	83	10·2	71	57·7	77	3·7	73	57·0	78	8·5	25	53·9	86	8·4	79	58·4	80	3·0
(*d*) Winter .	70	42·5	93	4·4	55	39·3	87	2·6	51	39·8	87	5·9	33	38·9	91	7·1	52	38·5	86	7·0
Total .	214	51·0	83	21·4	180	49·2	78	17·2	176	50·2	77	25·9	95	47·0	84	32·0	161	49·4	82	20·7

	1862.				1863.				1864.				1865.			
	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.
January .	17	° 39·0	85	1·9	20	° 41·8	85	2·6	6	° 36·5	82	0·9	14	° 36·3	89	3·3
February .	6	41·1	84	0·5	21	42·1	86	0·6	13	36·0	88	0·8	18	36·6	83	1·9
March . .	17	43·1	86	3·7	15	43·9	78	0·7	9	41·3	83	2·7	3	36·6	82	0·9
Apri . .	4	48 4	81	2·8	4	49·1	78	0·4	14	48·2	74	0·7	6	52·3	73	0·4
May . . .	5	55·4	84	2·8	9	52·0	78	1·3	11	53·8	73	1·9	5	56·1	73	4·4
June. . .	21	56·3	77	1·8	8	58·1	75	3·9	12	57·4	72	0·9	27	60·2	70	2·4
July . . .	21	59·1	78	1·7	14	60·8	72	0·9	18	61·8	70	0·3	26	63·8	72	2·3
August . .	14	59·5	81	3·0	12	61·9	74	1·8	51	59·6	65	1·4	34	59·9	80	4·0
September.	16	57·7	83	1·6	22	53·7	77	3·2	42	56·9	77	2·8	52	63·9	76	0·2
October .	31	51·8	89	4·0	24	51·6	87	1·7	39	50·5	78	1·1	85	51·4	87	5·9
November .	34	39·8	92	1·0	17	45·7	88	1·8	24	42·0	86	2·6	128	45·0	88	2·3
December .	18	43·6	88	1·6	9	43·2	83	1·1	10	38·5	86	0·6	106	43·5	90	0·6
(*a*) Spring .	27	44·2	83	7·0	40	45·0	80	1·7	36	41·8	81	4·2	27	41·8	79	3·2
(*b*) Summer	47	56·9	79	6·3	31	56·9	75	6·1	41	57·6	71	2·1	58	60·0	71	9·1
(*c*) Autumn	61	56·3	84	8·6	58	55·7	79	6·7	132	55·6	73	5·3	171	61·7	81	10·1
(*d*) Winter .	69	40·8	88	4·5	46	43·5	85	5·5	40	39·0	84	4·1	266	41·6	89	6·2
Total .	204	49·5	83	26·4	175	50·2	82	20·0	249	48·5	77	15·7	522	51·2	80	28·6

(*a*) Spring—February, March, April. (*b*) Summer—May, June, July. (*c*) Autumn—August, September, October. (*d*) Winter—November, December, January.

Second, that of all the seasons autumn is the one in which Enteric Fever is most prevalent. In fourteen out of the seventeen years, the number of autumnal cases exceeded that of any other season. In one of the three remaining years, 1851, an equal number of cases occurred in the summer and autumn respectively. In the other two years, 1860 and 1862, the greatest number of cases occurred during the winter, exceeding the autumnal cases by sixteen. *Third*, that the disease is least of all prevalent in the spring. Excepting the years 1852, '56, '59, '60, '61, and '63, the least number of cases occurred in spring, and in all these exceptional years there were only thirty-four spring cases in excess of the summer ones.

It appears from these general facts, and from a little closer examination of the table, that Enteric Fever obtains its maximum development in the months of September, October, and November, declines slowly during the winter and spring, and reaching its minimum in May, then begins to increase progressively with the advance of summer.

Let us now go a step further, and endeavour to find out the conditions which, prevailing most in autumn, render this season most favourable to the existence of Enteric Fever.

Temperature.—In every year but 1852, the combined temperature of the autumn and winter was less than the combined temperature of spring and summer, and in this exceptional year the mean temperatures were as 50°·3 to 50°·9. And in every year but 1851, '54, '55, '56, and '61, the temperature of autumn was less than that of summer, and in no year did the autumnal temperature exceed that of summer more than 1°·1. Again, if we except the years 1851, '55, '56, and '61, October —the month when Enteric Fever is most rife—was cooler than May, when the disease is at its lowest ebb. It has been already stated that in five of the years, the number of cases occurring in the spring of those years was in excess of the number which happened in the warmer summers.

Apart from any other cause, it cannot, therefore, be concluded that temperature has any influence on the increase of Enteric Fever.

Rainfall.—In considering the influence of rain upon the quantity of Enteric Fever, attention must be given, not so much to the total yearly amount, as to the quantity which falls in each month. The average amount of rain for each of the seventeen years is 23·1 inches. If only one inch fall during a period of two months, that must be regarded as a season of drought.

Of these seventeen years the most rain, 34·4 inches, fell in 1852, and in this year there were 140 cases of Enteric Fever. Next stands the year 1860, when there was a rainfall of thirty-two inches, and only ninety-five cases—a total considerably smaller than that of any other year. But on further comparison we find that in 1860 the rain was not only abundant, but that each month had a due share, while in 1852 the still more abundant rain was unequally distributed throughout the year, the spring and early part of summer being unusually dry. If now, on the other hand, we regard the influence of drought, we find that the largest number of cases, 249, occurred in the driest year, 1864, when only 15·7 inches of rain fell. The next driest year was 1858; there were only 17·2 inches of rain in this year, and one-third of it fell in the summer months; the winter was the season of drought, and this was associated with an unusual increase of Enteric Fever in January.

With the same amount of rain in the years 1850 and 1854, there is a difference of ninety-one cases of fever, and this may be attributed to the inequality of the distribution of rain throughout the year, which may possibly be greater than is indicated in the table. Thus, for example, the Table is not sufficiently detailed to show that, in 1854, the inch of rain for June did not fall on the first day of that month, and the 1·7 inch on the last day of July, leaving a long interval of drought between—as may have been the case. Mr. Glaisher's laborious and valuable observations are deserving of more detailed study in the elucidation of these questions.

The dry winters of 1851, '53, '55, '58, and '62, were associated with an increase of Enteric Fever.

The opposite effects of drought and rain have been well illustrated during the present summer (1865). Towards the end of July there was a great want of rain, but from the 30th of this month and throughout August there was an unusual amount, large quantities having fallen almost every day. As shown by the admissions into the London Fever Hospital, Enteric Fever was very prevalent during the dry season, but after a fortnight's heavy rain its further progress received a sudden check, which continued until the effects of the succeeding drought became manifest.

It appears clearly from the foregoing observations that the absence of rain furnishes conditions most favourable to the increase of Enteric Fever; and since drought is necessarily associated with dryness of the air and exalted temperature, we must consider it, thus combined, as the one predisposing cause of Enteric Fever.

Other Atmospheric Conditions.—Too little is at present known respecting the influence of ozone in the production of disease; but as this body has been observed to be absent, or nearly so, from the air during the prevalence of cholera and other intestinal affections, the following general statements made by Dr. Moffatt (Chemical News, September 1861) may be borne in mind. The quantity of ozone varies according to the time of year, the direction of the wind, temperature, atmospheric pressure, and the presence of decomposing substances. Rain, a south wind, fall of the barometer, and increase of temperature, separately or combined, are associated with an increase of ozone, and the reverse conditions with its decrease. "Ozone periods terminate with increasing barometer readings, decrease of temperature, and wind from N. points of the compass." Ozone is most abundant in January, February, and March; less so in April, May, and June; and least of all in July, August, and September. "The greatest number of ozone days is in April, and the smallest in August and November. Whatever tends to a deflection in the direction of the wind leads to a corresponding result in ozone observations; and a town, chemical works, drains and cesspools, &c., deozonize the air, or wind passing over them" (page 167).

Change of Residence, &c.—Both Louis and Chomel have observed that the greater number of the patients who came under their treatment in Paris had resided there only a short time. But change of residence, apart from the excitement and fatigue, the irregularity of living, and the distress which very commonly attends it, can hardly be considered a predisposing cause of Enteric Fever. In the autumn of 1861 a case of Enteric Fever, which terminated fatally, came under my care in Paris. The patient, a robust, newly-married lady, had been resident there only a few weeks, but during the whole of this time she had voluntarily lived a life of daily excitement and fatigue; the digestive functions were deranged by an unusual diet and irregular mode of living, and to these causes the disease was probably attributable.

M. Chomel found that one-third of the 115 cases to which special inquiry was directed had been exposed to sudden cold, to want of food or bad diet, to excessive fatigue, to mental depression, and to debility produced by other diseases.

In his account of the outbreak of Enteric Fever in a garrison of 306 soldiers, Dr. Grossheim says: "It is difficult, if not impossible, to ascribe any deleterious influence to the food—all shared alike." He attributes the disease to the effect of military exercises in a changeable season, with night bivouac in the open air. (Edin. Med. Jour. vol. xlviii.)

(*b*) *Exciting Causes.—Contagion* is supposed by MM. Leuret, Bretonneau, Gendron, Dr. William Budd, and other physicians, to be the means whereby Enteric Fever is propagated. The following examples of the spread of the disease furnish the strongest proof that can perhaps be adduced in support of this view:—

Five persons are successively attacked with Enteric Fever in a certain house in Geneva. A sixth inhabitant of that town spent two nights with the third patient, soon contracted Enteric Fever, and died of it in the hospital. "At the autopsy, ulcerations of the ileum, and all the other lesions characteristic of dothinentery, were found." A clergyman who visited the third patient—a little girl—took the disease and died with all the symptoms of typhoid fever; his nurse was also attacked with typhoid fever, and died in the third week. A young lady also paid the third patient a visit, and rendered her some service in the sick-chamber, and this person soon fell ill with symptoms of typhoid fever, in another house, in which five other persons were subsequently attacked by the same disease. (M. Lombard, Gaz. Méd. 1839, p. 138: quoted by M. Piedvache, Mém. de l'Acad. de Méd. tome xv. p. 294.)

Dr. W. Budd had seventeen cases of Enteric Fever under his care in the hamlet of North Tawton, Devon, and during the prevalence of the disease three persons left the hamlet ill of the fever.

A went to Morchard and died there, and ten days after his death two of his children had the fever in the same house.

B also went to Morchard, and three cases of Enteric Fever afterwards occurred in the house where he lay ill.

C went to Chaffcombe, seven miles from North Tawton, and nine other cases of Enteric Fever appeared in the farmhouses to which he went. One of these nine left Chaffcombe and went four miles away, to Loosebeare, to be nursed. Several inmates of the house into which this patient was received, were subsequently attacked with Enteric Fever, and from this house the disease extended over the whole hamlet. An infected boy also left Chaffcombe, and took the fever to a cottage midway between Bow and North Tawton, and five persons subsequently fell ill of Enteric Fever in the house into which he was received, and in the adjoining one. Besides these there was no single case of the sort nearer to Chaffcombe than North Tawton. "There were twenty or thirty hamlets in the neighbourhood similar in all respects to Loosebeare. From the soil of all, through month after month of the same fine, dry, autumnal weather, human and other exuviæ exhaled into the air; and yet, while at Loosebeare a large proportion of the inhabitants were lying prostrate with intestinal fever, in not one of the exactly similar places was there a single case." (Dr. W. Budd, *Lancet*, July 9, 1859, p. 28.)

It is reasonable that those who have witnessed such instances as these should be fully persuaded that Enteric Fever is propagated by contagion. But there are many physicians, and amongst them those who have had the most extensive experience of Enteric Fever, who conclude either that the disease is destitute altogether of contagious properties, or only possesses them in a very slight degree.

That form of Enteric Fever which prevails continuously in London is certainly non-contagious. I have never had cause for the slightest suspicion of contagion in any case which has come under my observation, either in King's College Hospital or in the London Fever Hospital, and this generally is the experience of hospital physicians, both in London and in Paris.

Since writing the above, the following instance, in which there is evidently a strong probability of contagion, has come under my notice. In the present summer (1865), four members, A, B, C, D, of a family of six adult people, residing at St. Peter's Terrace, Notting Hill, were attacked with Enteric Fever. A and B had been residing at Ryde for a month. They returned to town with two other members of the family on the 26th of May. A suffered ever since his return with diarrhœa, which continued, and the symptoms of Enteric Fever became well developed. "On the 28th of June he had a severe attack of intestinal hæmorrhage, which recurred two or three times daily for several days; eventually he recovered." About the 7th of June, his sister, B, fell ill of the disease, and she died comatose on the 29th of June. On July 10th, C and D, who had not been absent from London, manifested symptoms of Enteric Fever, and were sent into

King's College Hospital, where they came under my colleague Dr. George Johnson's care. I saw them frequently. They had well-marked Enteric Fever, and are, at this present date (August 22d, 1865), slowly convalescing in the Twining ward. On the 8th of August, a nurse in attendance upon these patients was taken ill with the same disease, and now is under my colleague Dr. Beale's care, in the same ward, in a very precarious state.[1] This last patient, it must be observed, slept every night at St. John's House, situated at a distance from the hospital, and between it and the Thames; and a great many isolated cases of Enteric Fever have occurred in London during this month. All but the last patient came under the care of my friend Dr. Easton, of Connaught Square, and to him I am indebted for the particulars having reference to the period before B and C were admitted into the hospital. Dr. Easton carefully inquired into the sanitary condition of the house where the disease broke out. No cause could be found, and care was taken to mix the evacuations with Condy's fluid and dispose of them immediately they were passed. C and D had scarcely any communication with A and B.

In considering the question of contagion it must be granted that the occurrence of any number of cases simultaneously or successively in any given house or hamlet, can never prove the fact of contagion. The general conditions, moreover, favourable to the increase of the disease, are so common and wide-spread, that one who entertained limited views of the nature of the disease, might account for its propagation among the inhabitants of Geneva, above referred to, for example, by assuming—and not improbably—that the conditions favourable for the spontaneous development of Enteric Fever existed in each of the four or five houses in which the patients resided, and that the intercommunication which took place between them was a curious coincidence—each patient in reality being independently affected. If this explanation be thought satisfactory in the first instance adduced, it cannot be considered otherwise than unlikely and superfluous in the second, in which the general evidence of contagion appears to be complete. It is true that the conditions favourable for the outbreak of Enteric Fever appeared to exist equally in all these little hamlets; and if it had arisen in any or in all without the intervention of an infected person—and it must have so arisen in the first case—the disease in each individual might reasonably have been referred to a spontaneous origin.

M. Gendron himself was unable to account for the first cases in many localities, and M. Piedvache, after a careful examination of the whole question of contagion, "feels almost sure that typhoid fever, under some circumstances, declares itself at once and in sufficient numbers to constitute an epidemic, independently of contagion." (Mém. de l'Acad. de Méd., tome xv. p. 137.)

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[1] She died on the 25th of the above month, and I witnessed the post-mortem examination. There was extensive ulceration of Peyer's patches and enlargement of the solitary glands in the last three feet of the ileum, and these constituted the chief anatomical lesions.

We have now to inquire into the conditions with which Enteric Fever is immediately associated, and the mode of its spontaneous origin.

Spontaneous Origin.—The majority of the French physicians who witnessed the epidemics which occurred in France, from 1841 to 1846, "signalize, amongst the causes to which they attribute the manifestation of these epidemics, the following conditions:—The more or less immediate vicinity of stagnant waters, marshes, or bogs, from which, chiefly under the influence of summer heat, effluvia arise and spread over the people—effluvia which tend to produce affections of a periodic type; the presence of dunghills, often accumulated and allowed to remain for a long time upon a public way, before houses, or the single door of some wretched hut; wells of water, level with the ground, permeated with water infected by drains and dunghills; an infected pool existing in the midst of a commune and furnishing only a brackish, muddy, stinking water for the common drink of men and of animals who come to allay their thirst there; or springs, containing, it is true, a pure water and fit for drink, but disturbed by all kinds of animals who go there to drink and corrupt the water with their dung; or further, conduits of impure water, which become a source of infection to the houses near which they pass, or, discharging their contents upon the public ways, form puddles of stagnant water in streets badly kept and unprovided with suitable means for carrying off the fluid accumulations." (M. de Claubry, op. cit. p. 11, et seq.)

The occupants of a farmhouse are attacked by Enteric Fever, and the only discoverable cause is an overflowing cesspool, or a stagnant, offensive pond, or a low-lying fold-yard, covered with a thick bed of dung and other refuse matter, from which putrescent runnings sink and saturate the soil in which the well, supplying the house, is excavated.

The accumulated or pent-up sewerage of a town escapes into the subjacent soil within and about it, soaking into the wells and defiling the drinking water, and giving off filthy emanations into the air, and an outbreak of Enteric Fever follows.

A particular drain becomes obstructed, bad odours arise into the houses in communication therewith, and sicken their inhabitants, and Enteric Fever soon manifests itself.

Such are the almost constantly observed conditions with which Enteric Fever is associated, and we must therefore conclude that this disease has a spontaneous origin in putrescent matters—that these, when preserved from the purifying influences of air and water, generate a poison, which when admitted into the body produces Enteric Fever. Leaving for the present all speculations as to the nature of the poison thus generated, we will now proceed to inquire how the poison or poisons gain admission into the system—whether by the lungs or by the alimentary canal. The inquiry is an important one, and we must be careful to avoid the influence of partial views. If we except the most thoroughly-drained towns (such as London, where, perhaps, Enteric Fever attains its minimum development), wherever the air is vitiated by filthy odours, the subjacent soil is permeated by the putrescent source from which they arise, and the water is also contaminated. Hence, in many cases, it may be difficult to determine whether the poison were conveyed by the air or by the drinking water. Dr. Murchison, in his elaborate work on the Continued Fevers of Great Britain, endeavours to prove that Enteric Fever arises from "sewer emanations." "So far as we know," he says, "it is necessary for the production of the poison of Enteric Fever that the matter undergoing fermentation be either in a confined space as in a drain or sewer, or that it be in a state of stagnation. Free exposure to the atmosphere, or constant dilution in a running stream, may not only render the poison inoperative, but may altogether prevent its formation. A privy outside a house is much less dangerous than a badly appointed water-closet within." (Pp. 452, 453.) In the instances which he adduces to support this view nothing is said respecting the water-supply. Assuming that there were no sources of water within the precincts of the buildings, the outbreak of fever in the Westminster School—shortly after the exhalation, from a foul and neglected sewer, of a disagreeable stench, so powerful as to induce nausea—appears, pretty clearly, to have been due to the foul air. In outbreaks of disease amongst bodies of soldiers, school-children, &c. we must be prepared to look for the cause abroad as well as at home. A body of young cricketers, for example, may quench their thirst in some impure stream, and thus contract a disease, for which some apparent cause may readily be discovered at home. There can be little doubt that Enteric Fever is occasionally generated by exhalation from putrescent matters, but usually, we believe, they are ineffectual in the production of the disease. It has never yet been proved experimentally that Enteric Fever may be generated by emanations from decomposing animal and vegetable matters. Nor does it appear from the observations of those who have made special investigation in this direction, that individuals whose occupations require them to spend much of their time in an atmosphere thus contaminated, evince an unusual proclivity to Enteric Fever. Dr. Guy compared the past and present condition of ninety-six nightmen, with about the same number of bricklayers, labourers, and brickmakers, and after a most critical investigation, he says: "An examination of the tabulated results of his inquiries must convince the most sceptical that the health of scavengers is fully equal to that of the labouring man with whom they are compared." (Journal Statis. Soc., 1848, vol. ii. p. 79.) This agrees with the observations of M. Parent Du Chatelet and others on the health of men who work in sewers. In the country it is a common practice to empty the privies and spread their contents over the gardens or adjoining fields. The filthy odours are endured as a temporary inconvenience, but experience proves that the practice is a harmless one. In two instances I have known the inhabitants of a house to be exposed for months to the direct emanations—in the one case, of a large cesspool; in the other, of a common sewer. In both cases a leaden pipe, in communication with the water-closets, was carried down inside the house and thence into the sewer or cesspool. After several unsuccessful explorations it was discovered that rats had eaten a large hole in the pipe leading to the cesspool, and through this aperture the fœtid exhalations from its surface arose directly into one of the sitting-rooms and a passage of the building, and on hot and damp days the effluvia were intolerably offensive. The cesspool was emptied at this time, and I witnessed the process. The contents were semi-fluid, of a dirty-black colour, and the surface of the pool was covered with large bubbles of gas. Now, although several persons were continually exposed to filthy exhalations, in the one case for many months, and in the other for several years, none suffered from diarrhœa or Enteric Fever. Numerous examples of this kind teach us that we must not be too ready to attribute Enteric Fever to foul air, but that we should be prepared to acknowledge other means by which the disease may be introduced into the system. Of these we may consider two,—impure water and bad food.

The following instance is worthy, from the simplicity of the attendant conditions, of attentive consideration with reference to the spontaneous origin of Enteric Fever. An outbreak of Enteric Fever occurred at Stangenrod, "a village chiefly seated on a naked, exposed, isolated eminence. Trees, excepting young brushwood, are not seen in the neighbourhood, and there are but few marshes; a small stream has its source at some distance, but scarcely touches the extreme boundaries of the district. In several places are flats covered with broom and used as pasture grounds. The basis is mostly basaltic rock, covered with a layer of clay and loam mixed with sand and flint. The air is keen and pure, and the atmosphere rarely disturbed by clouds. The position of the town is healthy as to local influences, and not favourable to the production of endemic disorders, which, it appears, had never been observed in it. The inhabitants were poor and chiefly occupied in agriculture and pasturage; comfort and wealth were exceptions. The houses were small, low, dirty, and surrounded by dunghills. The general want of spring-water the inhabitants attempt to supply by collecting rain-water in bad reservoirs." (Dr. Ebel, Ed. Med. and Surg. Jour., 1837, vol. xlviii. p. 160.) The disease appeared at the close of July 1833, and terminated in April 1834. The spring of 1833 was dry, followed by a dry summer, with insufferable heat, continuing to autumn without rain, and with little wind (p. 187). 157 out of 318 inhabitants were attacked, and 19, or 12·1 per cent., died. Dr. Ebel believed that the chief cause of the disease consisted in "the entire want of good fresh water and the use of corrupted water. The inhabitants of the village used for domestic purposes rain-water obtained from marshes, and preserved in insufficient reservoirs until required" (p. 188).

In the latter part of 1859 a severe outbreak of Enteric Fever occurred at Bedford, and there was every reason to believe that it was due to fæcal matter soaking into the wells from the numerous cesspools of the town. The water from these wells was found to contain a large quantity of decaying animal matter, evidently derived from the sources alluded to. (Simon, Report to Privy Council, 1860.) Early in October 1847, "intestinal fever" broke out almost simultaneously in thirteen houses in a certain terrace at Clifton. The houses were far apart in the terrace, and there was little or no intercourse between their inmates. The inhabitants of these thirteen houses drew their drinking water from a well situated at one end of the terrace, and at the end of September it became evident from the taste and smell of the water from the pump, that it was tainted with the contents of the sewer. The remaining twenty-one houses were supplied with water from another source. (Dr. W. Budd, *Lancet*, 1859, p. 432.) Other instances of the direct association of Enteric Fever with defective drainage and contaminated well-water may be found in the Sixth Report to the Privy Council, 1863.

Rain, the natural preventive of Enteric Fever, may, under certain local conditions, be the means of diffusing it. This appears to have been the case at Festiniog.

The houses, in which the disease appeared, are situated on the slopes or bases of mountains on the sides of the valley stream. "The majority have no privies nor ash-pits, nor have their inhabitants access to places of this sort. The custom of the neighbourhood is to use the fields, or when house utensils are employed to empty them at a distance from the houses. Houses in a row are generally provided with one or more privies, with cesspools common to the row. The air is pure and the natural drainage good; the valley stream is exposed to constant contamination. The inhabitants stated that water for internal use is got from wells on the mountain side, presumably situate above any possible source of pollution" (see the Report to the Privy Council by my colleague, Dr. Buchanan; Sixth Report, 1863, pp. 787–8); but they acknowledge that they use the water of the stream in the valley for some domestic purposes, and it is obvious how readily this may be substituted for the purer, on all occasions, and particularly when the distant spring fails, and laziness or lack of time constrains the water carrier.

Under such conditions as the above, filth accumulates upon the surface during a dry season, and remains harmless there till drenching rain gradually washes it down into the common stream, which thus becomes continuously contaminated for weeks or months. Dr. Buchanan has pointed out these conditions, and is inclined to attribute the prevalence of the disease during an unusually *wet* autumn to water contamination, combined with exposure to cold and wet.

Impure water appears to be equally a cause of intermittent fever and its complications. The Inspector of Hospitals writes of Walcheren during the prevalence of the severe intermittent fever there: "The bottom of every canal that has communication with the sea, is thickly covered with an ooze, which, when the tide is out, emits most offensive and noisome effluvia; every ditch is filled with water which is loaded with animal and vegetable substances in a state of putrefaction; and the whole island is so flat, and so near the level of the sea, that a large proportion of it is little better than a swamp; there is scarcely a place where water of a tolerably good quality can be procured." Sir John Pringle mentions "that the men-of-war which lay all the time at anchor in the channel, between South Beveland and Walcheren, even during the worst period of the distemper, were not affected with either flux or fever, but enjoyed the most perfect health." (Davis on the Walcheren Fever, 8vo. 1810, p. 15.) These sailors were doubtless provided with a supply of good water.

The inhabitants of a marsh, seated in a basin of clay, or level with the bed of a river, must of necessity drink water contaminated by their excretions and other impure matters, if the water be derived from the marsh itself, and the drier the season the more concentrated the poisonous impurity.

Dr. W. Budd most strongly insists that the essence of Enteric Fever is contained in the alvine dejections of the patient, but we cannot adduce any facts recorded by himself that give material support to this view, and our own observations lead us to the conclusion that the intestinal discharges do not contain any *volatile* poison which is capable of generating Enteric Fever.

Food in an incipient stage of putrefaction is also capable of generating symptoms and intestinal lesions apparently identical with those of Enteric Fever.

Dr. Kerner of Weinsberg has collected 135 observations of poisoning from eating a certain kind of smoked puddings, which he regards as putrid food. They are chiefly composed of animal matters, and have a putrid savour and odour. Post-mortem examinations often revealed traces of inflammation of the œsophagus and pharynx; gangrenous patches of the stomach; intestines inflamed in divers places, or even gangrenous in part; lungs strewn with black spots, or hepatized. (Orfila, vol. ii. p. 636, 1843.) Dr. Schumann records the symptoms and post-mortem appearances produced by eating similar food. The symptoms were those of Enteric Fever, *plus* inflammation of the pharynx, œsophagus, and larynx. Post-mortem examinations revealed inflammation of the intestinal canal: "L'intestin grêle, quelquefois très distendu par les gaz, présenti des traces d'inflammation très intense, et souvent des plaques gangréneuses." Lungs gorged with blood. (Archives Générales de Méd. tome xxii.)

Granting that Enteric Fever may be produced by the ingestion of putrid animal substances, we shall be at no loss to find a cause for the origin of many of the isolated cases which occur, for such may exist in every household. There is nothing, for example, more essentially putrid than the *decomposed cheese* with which many persons habitually indulge their appetites, and persons unaccustomed to such food can hardly be supposed to partake of it with impunity. It is a matter of common experience that an *egg* will sometimes produce vomiting and purging.

With regard to the identity and nature of the poisonous agent or agents which produce Enteric Fever, we know nothing. It has never been demonstrated that any particular gaseous body can induce the lesions found after death from Enteric Fever. There are, however, both mineral and vegetable substances which, when introduced into the stomach, produce symptoms and morbid changes, if not identical with those of Enteric Fever, at the least hard to be distinguished from them. Thus, to take vegetable substances:—Twenty-four hours after the ingestion of *poisonous mushrooms*, the members of a family were taken with nausea, and the next day frequent vomitings of bilious matters and abdominal pains, of which symptoms three children died. The other members had insupportable pains in the stomach, loins, and abdomen, meteorism, retention of urine, tenesmus, glairy sanguinolent stools, vomiting, and great thirst. On the fifth day, shivering, collapse, and death.

The stomach and intestines were found covered with whitish or yellowish mucus, the valvulæ conniventes and mucous follicles generally very prominent. (C. P. Galtier, Traité de Toxicologie, tome ii. pp. 564-569.)

The effects of *colchicum* still more closely resemble those of Enteric Fever. Two young women took each about ℥iv. of tincture of colchicum. Both died of violent gastro-intestinal inflammation. The viscera of one were tied up and sent away for analysis. In the other, who died twenty-eight hours after taking the poison, the lungs were healthy, stomach distended by gas, and the mucous membrane evidently softened. Throughout the whole extent of the intestine the muciparous follicles were of the size of millet seeds, and in the inferior third of the ileum the plates resulting from the agglomeration of the mucous crypts were also much developed and of a violet colour. (Galtier, loc. cit. p. 322.)

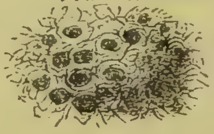
In poisoning by *Cicuta virosa* similar symptoms and swelling of the glands of the intestine are produced.

Has the poison which so frequently generates Enteric Fever any relation to the elaborated narcotico-acrid principles of these or other plants? If so, is it generated in the process of decomposition, and is it possible to obtain it by lixiviation of the impure soil, or by precipitation from the contaminated water? Or again, is the poisonous agent merely a common product of decomposition, and destitute, like that common contagious sanies which so readily induces phlebitis or erysipelas, of any specific character? Such are the speculations into which further inquiries respecting the origin of the disease lead us.

Returning now to the facts above detailed, we may conclude, (1) that refuse animal and vegetable matters, if allowed to accumulate and decompose in seasons of drought, generate a poison which, if not washed away or diluted by sufficient rain, rises into the air, or becomes diffused in the water; and which, when introduced into the body by these media, may produce Enteric Fever; (2) that food or water rendered impure under conditions less general, may also be the means of inducing the same disease; (3) that there can be little doubt that the usual symptoms and post-mortem appearances of Enteric Fever may arise during the progress of several other acute diseases as a consequence of a general inflammatory condition.

DIAGNOSIS.—The indications of inflammation and ulceration of Peyer's patches are the following:—Pain and tenderness in the right iliac fossa; general derangement of the alimentary canal, accompanied by persistent diarrhœa; light ochre-coloured watery stools; hectic fever; and the eruption of rose-coloured papules. If these symptoms be associated with any febrile condition or complication whatever, we may be sure of the presence of enteric inflammation. If they are absent, the case may be one of commencing pneumonia, erysipelas, hernia, puerperal fever. But each or all of the symptoms of enteric inflammation may be latent, and our attention may, therefore, be altogether called away from the abdomen to the head or the chest. In the absence of diarrhœa, the other characteristic symptoms being present, we may assume, generally, that the intestinal glands are simply inflamed and swollen; the presence of the pale, watery, flocculent stools, on the other hand, may be taken as indicative of the ulcerative stage. Diarrhœa alone, or even associated with a febrile state, does not furnish conclusive evidence of intestinal lesion; but there is one unmistakeable evidence occasionally present, from which, in the absence of every other symptom, we may positively declare that the agminated glands of the ileum are in a state of sloughing inflammation; it is the presence of a fragment of a disintegrated Peyer's gland in the stools. If the flocculent debris, when examined under water by the aid of a pocket lens, present a number of minute, closely-placed follicular depressions with minute circular orifices, loosely embedded in a ragged fibrous stroma (fig. 8), we have direct and positive evidence of the nature and progress of the disease. Even while the pale stools are still solid, a shreddy fragment presenting these unequivocal characters may be found adherent to their surface. To apply the test, the stools should be strained through a little cap net, the matters arrested thereby should then be washed, floated in clear water, and examined with a common pocket lens. The structure of the agminated gland is quite distinct from that of any other tissue in the body, not even excepting the thyroid gland and tonsils. Cellular vegetable structures, such as the rind of an orange, may be mistaken for the intestinal slough, but these are distinguished by their softer and more pulpy texture, and by their vegetable qualities. If there be much fever, with headache and delirium before the abdominal symptoms are developed, Enteric Fever may be mistaken for several other acute diseases, such as scarlatina, variola, typhus. In the first of these diseases we have the same bright cornea and flushed face, and the same lively character of the delirium. When characteristic symp-

FIG. 8. natl size.



toms are absent, we must suspend our diagnosis for a day or two, using at the same time precautionary measures against contagion. If the rash and sore throat of *scarlatina* be but slightly developed, or if the rash have receded, the diagnosis will be difficult and unsatisfactory.

If, in the accession of *variola*, the severe lumbar pain be absent, and the eruption make its first appearance as a few isolated papules upon the chest and abdomen, the case may be doubtful for a few hours, but the hardness of the variolous papule will speedily remove all doubt.

Chronic tubercular peritonitis presents many of the symptoms of Enteric Fever. Hectic flush, pinched features, abdominal pain, tenderness, gurgling, and diarrhœa are common to both diseases. But in tubercular peritonitis the tongue is clean and moist, there is no eruption, and frequently the abdomen is distended with fluid effusion; moreover, there may be evidence of tubercular deposit in the lungs.

Tubercular ulceration of the intestines, with tubercular deposit of the mesenteric glands, is a condition frequently mistaken for Enteric Fever. Two of M. Louis' cases (Obs. xliii. xliv. vol. ii.), given as examples of "latent typhoid fever," are instances of general tuberculosis simultaneously affecting the lungs and the intestines. I have already included another case (see case 12) recorded by this author (Obs. xxxiv.) under Tuberculosis, on account of the co-existence of miliary tubercles in the lungs and ulceration of the glands of the ileum, regarding the lesions in the chest and abdomen as simultaneous manifestations of the same pathological condition. If the lungs had presented evidence of only common inflammation, there would have been no reason for distinguishing this case from Enteric Fever. Indeed, the distinction between acute phthisis simultaneously affecting the lungs and intestines, and Enteric Fever, will more often turn upon the difference between tubercular and simple pneumonia than upon a difference in the character of the intestinal lesions. Inasmuch as the lungs are so commonly affected with inflammation in Enteric Fever, and the tubercular ulceration induces all the more prominent symptoms of Enteric Fever,—such as hectic, abdominal tenderness, tympanites, diarrhœa, and even intestinal hæmorrhage,—the diagnosis is often exceedingly difficult. If the patient have been long declining in health, with emaciation, sweating, cough, and expectoration, and we find dulness at either apex of the lungs, gurgling, or amphoric breathing, we may set down the case to be one of phthisis. Doubtless, it is possible for tubercle to be deposited nowhere else in the body but in the solitary and agminated glands of the intestine, and in the neighbouring mesenteric glands; and, in such a case, the diagnosis between the two diseases, hardly or not at all to be obtained during life, would turn upon the distinction between tubercular deposit and ulceration, and the nature of the inflammatory process and ulceration in Enteric Fever. I have examined the intestinal lesions of individuals in whom, after death, the lungs have been found consolidated with tubercles, and excavated into ragged cavities at their superior parts, side by side with the corresponding abdominal lesions of Enteric Fever. In the early period of the tubercular disease, I have been unable to detect any difference in the morbid phenomena; both diseases fall with greatest severity upon the solitary and agminated glands of the lower third of the ileum, and the appearance of the ulcers is similar, except that in Enteric Fever the inflammatory action and swelling is usually greater; in both diseases the contiguous mesenteric glands have the same violet colour, soft consistence, and increased size. The diseased parts offer the same microscopical appearances; but the corpuscles of the inflamed glands within and without the intestine are a little smaller in Tuberculosis than in Enteric Fever. In the advanced period of the tubercular disease, however, the ulcers are very distinct from those of Enteric Fever; they have an irregularly circular, thick, often indurated, elevated, angry-looking border, inclosing, and here and there continuous with, large interrupted irritable granulations, between which little yellow masses of tubercular matter, firmly attached to the base of the ulcer, are to be seen. In chronic cases, moreover, and before ulceration has begun, the swollen intestinal and mesenteric glands are free from inflammation, and the tubercular matter within them has a white, opaque appearance. It frequently becomes hard, gritty, and cretaceous, and sometimes forms compact stony calculi.

The disease, however, with which Enteric Fever is most frequently confused, is *typhus*.[1] In our own country, Willis, Sydenham, Huxham and other acute observers, in every generation, have recognised and asserted the distinction between these two fevers, but a general confusion of the diseases has, nevertheless, prevailed amongst the members of the profession, until within some twenty years ago, and we are greatly indebted to Dr. A. P. Stewart of the Middlesex Hospital, and to Sir W. Jenner, for reasserting and proving that a distinction really exists, and for rendering the diagnosis between the two diseases more clear and distinct. What these observers have done for England, Dr. H. C. Lombard of Geneva, Messrs. Gerhard and Pennock of Philadelphia, Hildenbrand and Griesinger in Germany, and Forget, Godélier, Barralier, and others in France, have done for their countries.

Still, there are physicians who, either from partial views, or from insufficient experience of the two diseases, do not recognise any specific difference between them, and regard the abdominal lesion merely as a complication of typhus. From what has been said on the associated pathology of Enteric Fever, it may, perhaps, be argued that the abdominal lesion, which is assumed to be characteristic of Enteric Fever, is nothing more than a local result of a common febrile disorder of the system; yet it is remarkable that lesions of Peyer's patches, which may sometimes be found in every other acute disease, are never, I believe, found in typhus, in which the febrile condition is both very general and very prolonged. I have examined the intestines in a great many cases of typhus, with the exclusive view of detecting, if possible, some participation of Peyer's patches in the general inflammation, but have always failed to detect either swelling or ulceration.

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[1] See article Typhus Fever, p. 444.

Case 20.—Three months ago, a woman, aged 30, and her little daughter, came under my care, with well-marked symptoms of typhus. The hot dusky skin of both patients was maculated with a distinct mulberry, typhus rash, which in the mother was petechial on the twelfth day of the disease. On the following two days the rash was still more plainly marked, and the case offered a good example of "spotted fever." There were no gastric or intestinal symptoms; sordes formed on the teeth, and the tongue was dry and brown; the eyes were dull and suffused; the mind heavy and confused—in short, all the symptoms of typhus were most distinctly marked. On the fifteenth day the bowels became loose, and a considerable quantity of blood was passed *per anum* the same day. At 11 A.M. on the sixteenth day intestinal hæmorrhage was again declared by the passage of much clotted blood. The hæmorrhage continued during the day, and at one time the bed was saturated with it and it ran down upon the floor. She sank and died at 5·45 P.M. on the same day. I was induced to re-examine carefully the skin of her little daughter who lay in an adjoining bed. A dark typhus rash was still out upon the chest and abdomen, and there were no traces of abdominal mischief.

On post-mortem examination of the body of the mother, which was well developed and well nourished, I found the small intestine perfectly healthy and pale; every Peyer's gland was also pale and healthy, and no solitary gland was either enlarged or prominent. The mesenteric glands and spleen were normal in size and appearance. In the cæcum, an inch and a half beneath the margin of the ileo-cæcal valve, I found a circular ulcer of the mucous membrane a quarter of an inch in diameter; the surface was greyish-white, like an aphthous ulcer of the mouth; the margin was slightly excavated, slightly raised, and vascular. On the opposite wall of the cæcum, a little higher up, where it becomes ascending colon, there was another much larger ulcer; it was an inch and a half long by half an inch wide, and it lay across the bowel; the surface was irregularly excavated, clean, and pale; the margins irregular and sharp; it extended below the mucous membrane, and lay in the thickened areolar tissue. In the transverse colon there were eight other small ulcers, resembling the first one, but deeper, and with margins more sharply cut and vascular. Excepting the largest, all the other ulcers were placed over one or other of the bands of longitudinal muscular fibres. Seven were arranged in a row upon one of them, at distances of about an inch. The intervening mucous membrane was congested, but not swollen; the ulcers were plainly the source of the hæmorrhage.

Such are the characters and situation of the intestinal lesions in typhus fever when they occur, but they are very uncommon. They do not appear to commence, like tubercular ulcers and the ulcers of Enteric Fever, in the solitary and agminated glands. In the case last described, the solitary glands contiguous to the ulcers were perfectly healthy. If we now compare the general symptoms of the two diseases, we shall find equally well-marked differences. The countenance in typhus is dusky, or suffused with a dusky blush, which spreads continuously over the face, neck, and shoulders; the eyes are injected and the pupils contracted; the expression is heavy, the intellect dull, and the delirium generally quiet. In Enteric Fever, the expression is bright, the pupils are dilated, and the hectic blush patchy, and limited to the cheeks; the delirium is often intermittent, becoming worse at night, and disappearing during the day. Its character is more lively than that of typhus. The eruption in typhus is a mere indistinct passive congestion, soon becoming a minute extravasation, and forms no distinct elevation. The rose spots of Enteric Fever are raised inflammatory papules. Diarrhœa, often profuse, frequently accompanies typhus, but the stools are always dark; the bile is thick and dark coloured. The patient has no peculiar odour in Enteric Fever, but in typhus the disgusting exhalations from the skin are so strong and peculiar, that we may often diagnose typhus by means of the nose alone. Typhus runs a regular course, and is terminated in the third week. Enteric Fever has no regular course, no certain date of termination. Typhus kills by coma or congestion of the lungs, Enteric Fever by asthenia, pneumonia, diarrhœa, hæmorrhage, or perforation —very rarely by coma.

PROGNOSIS.—The prognosis in Enteric Fever must be formed with extreme caution; the worst accidents of the disease sometimes occur when all appears to be going on well. The mortality, however, is not great. Out of 18,602 cases, there were 3,447 deaths, or one in every 5·4. (Murchison, p. 529.) A pulse continuously frequent, and much hectic or obstinate diarrhœa, are very unfavourable symptoms. Hæmorrhage, to any considerable extent, associated with tympanites, and occuring at a late period of the disease, is an equally unfavourable symptom. If there be excessive tympanites and abdominal pain, there is little hope. A fatal termination must also be anticipated if the stupor or delirium becomes continuous, and associated with muscular twitchings. The delirium sometimes assumes the traumatic character, or the patient becomes obstinate as well as stupid; such features also forebode an unfavourable issue.

On the other hand, we hail a fall in the temperature of the skin and the appearance of a copious eruption of sudamina over the abdomen and chest as most favourable indications.

TREATMENT.—The early recognition of enteric disease is of the utmost importance, for its progress is frequently so very insidious that many patients go about their usual occupations at a time when Peyer's glands would present very grave lesions (*e.g.* case 4), and complaining of nothing more than loss of appetite and debility. Our suspicions must be on the alert in such cases, and, before all things, we must ascertain the condition of the alvine secretions. Long before diarrhœa sets in, the well-formed fæces may be observed to be of a light-yellow or ochre colour—a condition in which they may exist in the absence of diarrhœa, but at a time when the intestine may be gravely ulcerated. The indications in the early period of the disease are to relieve internal congestion, and to revive the function of the liver. If the bowels be confined, a dose of castor oil, or of compound rhubarb powder should be given. If the skin be inactive, the hot bath should be used, and a diaphoretic, composed of aromatic spirit of ammonia and acetate of ammonia, prescribed. With the view of arousing the liver to activity, I have given the following every three or four hours, for two or three days, until some improvement was manifest in the condition of the alvine secretions. When necessary, I have continued the administration of the mercurial until its constitutional effects—slight tenderness of the gums and fœtor of the breath—began to appear.

℞ Hydrargyri cum cretâ, gr. iij.
Pulvis cretæ aromaticæ cum opio, gr. v.
Sodæ bicarbonatis, gr. xx. Misce, et fiat pulvis.

If this relaxes the bowels, I have combined it with a little more of the chalk and opium powder, or suspended its use altogether, and trusted to infrictions of mercurial ointment into the groins or armpits. I have employed this treatment in all cases which have come under my care in the early period of the disease, before diarrhœa had become severe, and have reason to believe that marked benefit has resulted therefrom, for the subsequent course of the disease in these cases has been uniformly mild. Calomel should be avoided; its action is too irritant. Saline purgatives and the vegetable cathartics must never be employed in the treatment of this disease at any period. A single dose of jalap, scammony, and the like, may suffice to develop the worst features of the disease. If, at an early period, we succeed in producing a flow of healthy bile with moderate action of the bowels, we may arrest further progress of the disease and restore the appetite. If there be diarrhœa and sickness from the commencement, we may prescribe an ounce of chalk mixture with ten grains of subnitrate of bismuth and five minims each of tincture of opium and dilute hydrocyanic acid, every two or three hours. This usually succeeds in allaying the vomiting. For simple vomiting of sour bilious fluid, a mixture, containing thirty grains of bicarbonate of soda and five minims of dilute hydrocyanic acid is very serviceable. The acid condition of the secretions in the upper portion of the alimentary canal, and the deficiency or total absence of alkali from the bile, are facts to be borne in mind in the treatment of Enteric Fever.

If the disease exhibit an intermittent character, quinine, gr. v. to x. may be given every evening. In the absence of cerebral or gastric disturbance, quinine is a most valuable remedy for subduing the evening exacerbations of fever so frequently present. Grains iij. to x.

may be given in a glass of water, with this view, every or every other day at noon.

In the further progress of the disease, our treatment will have almost exclusive reference to the abdominal lesion.

Diarrhœa must be restrained, some physicians think checked, if possible, altogether. My own experience, however, leads me to believe that moderate diarrhœa in the early period of the disease is beneficial in some cases, and more especially in those in which delirium appears early. In the congested condition of the abdominal viscera which exists in Enteric Fever, one or two liquid stools in the course of the twenty-four hours doubtless acts beneficially in all cases, and our object in the early period of the disease should be to keep the diarrhœa within moderate limits, rather than stop it altogether. In the early period, chalk and bismuth, with catechu and opium, is usually all that is needed to check the diarrhœa and allay irritation. As soon, however, as the diarrhœa becomes excessive, or we have reason to suspect ulceration, stronger astringents must be given. Some physicians use sulphuric acid with opium—℞ Acidi sulphurici diluti, ♏xxx.; tincturæ opii, ♏x.; decocti cinchonæ, ℥iss.; fiat haustus, quartis horis sumendus. We prefer the styptic salts, having found them much more efficacious: indeed, the acid mixture often increases the purging and pain. Acetate of lead, nitrate of silver, and sulphate of copper are employed. The first may be given in the form of mixture—℞ Plumbi acetatis, gr. iij.–v.; acidi acetici, ♏iij.; morphiæ acetatis, gr. ⅛th; aquæ cinnamomi, ℥iss.; fiat haustus, quartis horis sumendus. Acetate of lead is a very suitable and efficacious remedy, but its continued use in Enteric Fever should be avoided, as it may subsequently affect the system injuriously. Dr. Tweedie and M. Trousseau speak in high terms of nitrate of silver. It may be given combined with a grain or two of compound soap-pill in doses of a quarter of a grain to one grain, every three or four hours. Dr. Tweedie says: "I have prescribed it extensively in Enteric Fever, and continued its use for a considerable time, and have never witnessed any approach to discolouration of the skin." (Lects. on Continued Fevers, p. 233.) Of all medicines, we consider sulphate of copper to be the most efficacious in restraining the diarrhœa of Enteric Fever. We may give it in quarter grain doses, combined with two grains of compound soap-pill, to be taken every two, three, or four hours. If need be, the dose may be increased to a grain, a day or two afterwards. For children, it may be prescribed in doses of the eighth or sixth of a grain. If too large a dose be given at first, it may excite vomiting. In small doses we have often prescribed it when there has been considerable irritability of the stomach, in which cases it appears to act as a sedative. Alum, catechu, tannic and gallic acids, krameria, hæmatoxylon, &c. are of comparatively little value in the treatment of Enteric Fever.

Starch and opium enemata—(Mucilaginis amyli, ℥iv.; tincturæ opii, ♏xv.–xxx.; fiat enema, nocte vel nocte maneque injiciendum)—are of great value in allaying that irritability of the lower bowel which often induces purging. When enemata cannot be retained, we may still use suppositories. (Pilulæ saponis compositæ, gr. v. to gr. x.)

Abdominal pain and tenderness.—The disease being localized in the right iliac region, we must direct our remedies to this part. Hot sedative fomentations, turpentine stupes, or poultices containing an admixture of mustard, should be frequently applied to the abdomen. If there be much tenderness four or six leeches should be applied, partly to the right iliac region and partly around the orifice of the bowel. Leeching of the anus is the most effectual mode of relieving the intestinal congestion. If the pain be great, an occasional full dose of opium will be needed.

Tympanites.—If there be any increased fulness of the abdomen, a flannel or linen bandage should be placed around it. In commencing tympanites we regard this as a very important part of the treatment, as it at once diminishes the congestion of the inflamed part, and prevents injurious distension. It also gives support to the painful abdomen in the process of respiration. Folds of wet cloths may be interposed between the bandage and the abdomen.

If the tympanites be considerable it becomes a most distressing symptom, and the life of the patient is in great danger from distension of the diseased and attenuated bowel; laceration of its ulcerated coats being imminent so long as the distension continues. To relieve this painful and dangerous condition, turpentine stupes should be applied over the whole of the abdomen, and gentle support given by means of a thin flannel bandage. An assafœtida enema (℥xij. ad ℥xx. enematis assafœtidæ P.B.) often gives much relief. If we should fail, however, to cause expulsion of the air by this means, a long elastic tube with wide side openings may be passed into the colon and retained there at intervals. Dr. Tweedie speaks well of the use of the stomach pump, per rectum, in the relief of this condition. "I have applied it," he says, "in some cases with happy effects, and withdrawn the accumulated air which may be passed through the lower tube of the stomach pump into a basin containing water." (Op. cit. p. 237.) Oil of turpentine (♏x.–xx.), or oil of rue (♏iii.–v.), combined with opium and given by mouth, are often serviceable in the relief of pain and flatulent distension.

Intestinal Hæmorrhage.—Moderate capillary hæmorrhage from the general mucous surface of the bowel must be regarded as beneficial, and we should employ no means to check it, but if the blood be clotted, in large quantity, and unmixed with mucus, we must fear the erosion of a large vessel, and treat for such an accident very promptly. A bladder of ice bandaged upon the right side of the abdomen, and the internal administration of gallic acid, solution of perchloride of iron, acetate of lead, or turpentine, are the most hopeful means of arresting it. Sulphate of copper in combination with soap-pill is a very valuable remedy in this condition also, and one upon which we are inclined to place most reliance. If the patient have been previously taking the copper salt, the dose may be increased at once to one or two grains. Turpentine in doses of ten or fifteen minims given every half-hour or hour, is often effectual in stopping the hæmorrhage, and is especially useful in cases where there is a tendency to syncope. The solution of perchloride of iron of the British Pharmacopœia is a very valuable remedy for intestinal hæmorrhage ; ♏xx.–xxx. in a wine-glassful of water may be given by mouth every two or three hours. If the hæmorrhage be slight and the arterial action much excited, ♏xv. tincturæ digitalis, with ♏xxx. tincturæ ferri perchloridi, ℥iss. aquæ menthæ piperitæ, may be given every four hours. This may be administered alone or in combination with thirty minims of dilute sulphuric acid. If we fail to arrest the hæmorrhage by these means, the bowel may be injected with one or other of the following enemata. ℞ Plumbi acetatis, gr. x. ; acidi acetici, ♏x. ; morphiæ acetatis, gr. $\frac{1}{2}$; aquæ tepidæ, ℥iv. ; misce. ℞ Liquoris ferri perchloridi, ♏xv. ; morphiæ hydrochloratis, gr. $\frac{1}{2}$; aquæ tepidæ, ℥iv. ; misce.

Cerebral Symptoms.—The indications in the treatment of cerebral symptoms are to relieve congestion and procure sleep. If there be much pain and heat of the head, cold water may be applied as an occasional douche, a gallon being poured in a gentle stream upon the head as often as the heat becomes excessive. Rags wetted with water, or spirit and water, may be applied in the intervals. If this treatment fail to restrain the vascular excitement, a few leeches should be applied behind the ears, or a blister upon the nape of the neck. As often as they are required, full doses of opium should be given to procure sleep. In the majority of cases the cerebral affection is mild and requires no direct treatment, and the sedatives given to relieve the abdominal symptoms are usually sufficient to calm the nervous irritability and procure sleep. When the pulse is fast and feeble and there is pulmonary inflammation, we must be careful to avoid large doses of opium. In some cases the delirium makes the patient obstinate, and he persists in refusing food and drink and keeps the teeth firmly clenched. In such a case, with diarrhœa present or impending, we cannot feed *per rectum;* we must therefore gag the patient and use the stomach-pump.

In such a state, too, we should daily examine the pubic region. Now and then we are painfully reminded of the negligence of those in close attention upon the patient, by discovering, after death, the bladder distended almost to the umbilicus, and with its attenuated coats inflamed and softened.

Pulmonary Symptoms.—Bearing in mind the frequency of pulmonary complications, we should carefully regard the breathing, and occasionally examine the chest. If pain and crepitation be developed in any part of the chest, a blister should be applied and mercurial infrictions used. Cough and bronchial dyspnœa may be treated with ipecacuanha and senega and the application of mustard poultices and turpentine stupes.

Food.—While we are thus combating the disease, the most unwearied attention must be given to the support of the patient. The blood impoverishes, and the body emaciates very rapidly, and our endeavour must be to introduce such food into the stomach as will be most easily digested. All nourishment must be given in a fluid or pultaceous form. Eggs, milk, vermicelli, arrowroot, or ground rice, beef-tea, gelatin—alone, or in various combinations—will be the most appropriate articles of diet. The eggs must be given in the form of emulsion in a little wine whey, tea, or cocoa. Two or three should be given daily. Milk-arrowroot, containing a little brandy, is a very appropriate nutriment. The beef-tea may be thickened with well-stewed vermicelli, or isinglass. Small quantities of food should be given at a time, and repeated every one or two hours.

Stimulants, in any considerable quantity, are not needed in the early period of the disease. When required they should be given well diluted. A few ounces of wine in the form of wine whey, or dry port mixed with an equal quantity of water, may be given with a little sponge-cake at intervals. Effervescent wines must of course be avoided. If the heart's action be weak, or the patient tends to lapse into the typhous state, brandy may be freely given, carefully avoiding excess. The following general rules may be observed in the administration of alcoholic stimulants in this disease. As long as the pulse remains under 120 and retains moderate force, six to eight ounces of wine, or four ounces of brandy, given within twenty-four hours, will be sufficient. When the pulse ranges between 120 and 130, and is small, we may double these quantities; and if the heart does not respond to the stimulant after twelve hours, thrice the original amount may be given. The bulk and force of the pulse must be our chief guides, and if these notably fail from day to day, we must daily increase the quantity of the stimulant until the patient is supplied with as much as half an ounce every half-hour, always diluted with a little milk, tea, water, &c. When there is much hectic, and the pulse is small and sharp, strong stimulants often appear to increase the irritability of the system, and in such a case we should give them sparingly and in the early part of the day, trusting to a dose of quinine, with or without opium, according to circumstances, in the evening.

Excepting a little custard, solid food of all kind must be absolutely avoided, until a week after the diarrhœa has ceased, and the stools become solid. Then we may venture to order boiled fish with bread. A boiled egg, a little fish, or a ripe pear or plum, taken too early, will almost certainly bring back the diarrhœa with a complete relapse.

The patient must return very gradually to ordinary diet, and he should be directed to eat slowly and masticate the food thoroughly. At first boiled rice should be taken in place of potatoes.

Convalescence is sometimes very slow, and often retarded by the occasional recurrence of diarrhœa. The styptic should be continued a week after the stools have become solid. At first the bowels are usually constipated, and this condition we shall do well to maintain for days. Subsequently, it will be advisable to relieve the bowels occasionally by a dose of castor oil.

As soon as the digestive function is restored, we prescribe cod-liver oil as a supplement to the diet, in all cases where there is much emaciation, and if, as is rarely the case, the oil does not digest, we may direct it to be rubbed into the abdomen.

PROPHYLAXIS.—Sufficient evidence has been adduced to prove that Enteric Fever commonly arises from the retention of refuse animal and vegetable substances within an undrained, or imperfectly drained soil. If, therefore, the contamination of the soil be prevented by the construction of sufficiently inclined sewers with impermeable walls, and the inhabitants be provided with abundance of pure water, Enteric Fever may be expected to disappear almost entirely.

The requirements for the prevention of the disease are sufficiently simple, but they are not easily fulfilled in every place where living beings are congregated. Nature, indeed, has provided these sanitary conditions almost everywhere, and if man would be more mindful of them his life would be rarely sacrificed to Enteric Fever. A house built upon a hill-side, with its spring of pure water above the foundation, and its cesspool below it, would be free from this disease, as far as external conditions are concerned. But reverse the position of the cesspool and the spring, and the disease may appear at any time.

If the dwelling be built upon a low-lying flat, and there is no near spring or flowing stream, these two necessaries—a pump and a cesspool—must needs co-exist side by side. In such a case they should be as widely separated as possible, and the sides of the well should be thickly covered with concrete. Whenever the premises are small, and it can be so contrived, a water-closet should be provided, and the excretions carried in an impermeable drain to a distance from the pump. In towns and large villages both pumps and cesspools should be abolished, and every house provided with a water-closet in communication with a sewer. The water should be derived from a distant elevated spring or reservoir, preserved from contamination at its source, and conveyed in well-joined iron pipes to its destination. The soil should be well-drained, and during the continuance of dry weather the drains and sewers should be regularly flushed.

We cannot positively say that Enteric Fever arises from the ingestion of diseased meat, but there is a strong probability that it does sometimes originate in this cause. Whether this cause has been in operation during the present year (1865), when "contagious typhus" has been so generally prevalent amongst, and destructive of our horned cattle, there is no direct evidence to show; but it is remarkable that, coincidentally with the spread of the cattle disease, there has been a great increase of Enteric Fever. On turning to the Table at p. 401, it will appear that the number of cases admitted into the London Fever Hospital in 1865 is more than double that of every preceding year, and more than treble that of the majority. The only effectual way of preventing the admission of diseased meat into the markets would be to establish a limited number of slaughter-houses, where the animals previous to being slaughtered and afterwards, could be inspected by proper officers. The experience of the present severe epidemic of cattle disease has taught us, that, *after death*, it is exceedingly difficult and, to an inexperienced eye, impossible, to distinguish positively between the flesh of an animal which has died of contagious fever, and that of one slaughtered in perfect health. In the absence of that more general protection which is so urgently required, two precautions should be taken: first, flesh of a flabby consistence and of a dusky, dead hue should be avoided; and secondly, all meat should be so thoroughly cooked that the fibre is quite firm and free from juice, which, on exposure to the air, becomes red. In the treatment of the contagious variety of the disease, the ordinary precautions against contagion must be taken, viz. the isolation of the patient and the disinfection of everything that has had contact with him.

TYPHUS FEVER.

BY GEORGE BUCHANAN, M.D.

DEFINITION.—Typhus Fever is an acute specific disease, lasting from fourteen to twenty-one days, characterised by an eruption of its own that appears between the third and sixth day, eminently contagious, and forming strongly-marked epidemics.

Under the name "Typhus," the writer of one of the Hippocratic treatises describes a disease that agrees in its essential features with typhoid fever. But the term was not afterwards used to signify a special disease until the time of Sauvages, in whose nosology it is adapted to certain forms of continued fever, while the name Synochus is used for another class of cases. The disease as above defined, and now known as Typhus, has been separated from other forms of continued fever within the last thirty years.

Typhus Fever has received a multitude of names, almost every epidemic having added some fresh one. In English, Spotted Fever, Petechial Typhus, Epidemic or Contagious Fever, Putrid or Malignant Fever, Camp or Jail Fever, are samples of the names that have been conferred on the disease, from various considerations of its nature or cause.

ETIOLOGY.—The causes that predispose to Typhus may be considered as affecting the individual and the community.

In the individual, sex and age have no influence in determining an attack. In the writer's experience, at the London Fever Hospital, very nearly equal numbers of each sex, and persons of every age, from a fortnight to over eighty years, have been attacked. Upon the authority of death registers and hospital statistics, the statement is constantly made that Typhus attacks adults more than children; but the evidence furnished by these data is quite untrustworthy as showing the relative proclivity of different ages to an attack. Typhus, as it appears on the death registers, is indeed incomparably more frequent among adults than children; but that is because children rarely die of it, not because they are rarely attacked. And in hospital records a much greater proportion of adults than of children are seen to be admitted; but this is because of obvious domestic reasons, because of the slightness of the fever in children, and often because of the rules of the institution. When inquiry as to age is made to include every case of attack, children and adults are found to be quite equally susceptible; the actual incidence may even be observed to be strongly upon the young, partly because of their greater numbers, and partly because adults are frequently protected by previous attack.

Depressing mental influences, overwork, and anxiety, appear to be causes that render the system more liable to contagion. Fear of contagion is often alleged, and perhaps justly, to be another such cause. It is especially among persons of better rank of life that these influences have been observed to operate. Depressing bodily influences are, however, of far greater moment. With persons temperate and provided with sufficient food, the contagion of Typhus, even though intense, is usually resisted for some time; but with intemperate and ill-fed persons, contagion is received so readily, and so small a quantity of it produces an attack, that it is constantly difficult to find out whether in the particular case there has been any exposure to contagion at all. It is essentially among the poor underfed population of large towns that Typhus epidemics occur. Paupers and the class just above paupers are the chief, and except in intense epidemics almost the only, sufferers from the disease. People whose earnings enable them to get more than the bare necessaries of life from hand to mouth do not suffer from Typhus save in exceptional instances, and usually as a consequence of constant communication with the sick.

The most violent epidemics of Typhus have been among communities that were fed more badly than usual, either through social difficulties, or through failure of crops; special hardships in war, and in civil life, strikes, and commercial distress, have at different times determined an epidemic prevalence of the fever.

The experience of Ireland in 1818 and 1847 illustrates the influence of privation in predisposing a community to Typhus. In each of those years an epidemic of this fever prevailed (along with relapsing fever), as a consequence of the almost complete failure of the potato crop; and it is estimated that on each occasion an eighth part of the entire population was attacked. But it must not be supposed that serious epidemics of Typhus require exceptional destitution as a necessary condition of their occurrence. A marked instance to the contrary as afforded by a recent intense outbreak at Greenock, where circumstances of special commercial prosperity had (by causing exceptional overcrowding) conduced to the epidemic spread of Typhus.

The next predisposing cause of Typhus is probably the most important of all, and consists in the association of conditions known as overcrowding, crowd-poisoning, or ochlesis. These conditions are scarcely to be separated from each other, but may be enumerated as overcrowding of dwelling-houses upon too limited area, overcrowding of rooms by too many occupants, bad ventilation of streets and houses, domestic and personal dirtiness. It is to the operation of this series of conditions that the special incidence of Typhus upon the labouring population of large towns is to be ascribed.

Illustrations to the effect of crowding too many inhabited dwellings upon a limited area might be gathered in abundance from the experience of camps, where the superficial space per man has been less than in the densest cities with their tall houses, and where Typhus has carried off large proportions of many armies. But they may equally be drawn from the experience of civil life. The town of England which habitually has most Typhus, and in which the most serious epidemics occur, is Liverpool. Here the huddling together of houses with insufficient space around them is carried to a greater degree than in any other town in the kingdom. In Liverpool, a large number of the houses are built back to back, in unventilated courts, and the population is so dense that, in some districts, each person gets only eight square yards of superficial space. In these parts it is that fever specially flourishes, and in epidemic periods passes by none but those who are protected by previous attack. Glasgow is another instance of a town in which the packing together of houses reaches an extreme extent, and in which Typhus correspondingly prevails; its distribution following so exactly the degree of density of population in different parts of the town as to leave no doubt of the connexion between the disease and this condition.

Overcrowding of the interior of houses by too many occupants, with deficient ventilation of rooms, may be illustrated as a cause of Typhus by the experiences of the common lodging-houses of English towns. Before the regulation of these by law in 1851, dwellings of this class were in a state of most miserable filth and overcrowding. In London and Liverpool, especially, there is evidence that they were peculiarly infested with Typhus, far more cases of this fever occurring in them than among an equal population residing in poor tenements of another class. Since 1851, the number of fever cases in common lodging-houses has been accurately ascertained, under the same act of Parliament that has diminished their overcrowding and improved their cleanliness; and it is found that in some thousands of such houses in London, hardly any Typhus exists in non-epidemic times, and that in epidemic times they suffer now much less than other houses inhabited by the poor. In Liverpool, it was upon the overcrowded lodging-houses that the chief force of the epidemic of 1847 fell; the cases of Typhus that occurred in them being numbered by thousands. During the year 1863, when the fever again became epidemic, in a thousand regulated lodging-houses of Liverpool, only twenty-four cases occurred, a quite inappreciable fraction of the whole number of fever cases in the town.

Doubtless both sorts of overcrowding act chiefly by facilitating communication between the sick, and contagion from person to person. And hence comes one explanation of the different degrees in which overcrowding favours Typhus in town and country. In many country cottages, very considerable crowding of rooms, no ventilation, and habitual dirtiness exist as constant conditions, and yet Typhus is practically unknown.

Other predisposing causes of Typhus require brief consideration. Persons of all countries and races exposed to its influence contract Typhus with equal readiness. But the disease is essentially one of cold and temperate climates. Within such climates there is no country whose epidemics are accurately recorded, that does not suffer more or less from Typhus; and, on the other hand, there is no sufficient evidence that this fever occurs within the tropics. Of all countries, Great Britain and Ireland are the chief seats of Typhus, which occurs here more constantly from year to year, and with severer accessions of epidemic force than elsewhere.

In the United Kingdom, the large trading ports are especially prone to Typhus; but this must be ascribed less to their position on seaboard or river than to the greater communication they have with localities from which infection may be derived, and the extreme density of population in such places.

By season and meteorological influences Typhus is not known to be very much influenced. It is a very common, though not an invariable occurrence, that the last month or two of the year is the chosen period for an increase in the prevalence of Typhus. In the main this connexion appears to be established through the influence of cold upon the vital powers of the individual, and upon the social and domestic conditions of the poor community. People suffer more from scantiness of food and get less ventilation in their crowded rooms as winter sets in. Drought, again, has sometimes appeared, as recently in Bristol, to predispose to Typhus, through reducing the supply of the element necessary to cleanliness. Low elevation of site, again, is a condition that renders a place less easily purified by currents of air than if it lay higher, and may in some minute degree assist in the development of an epidemic. But to such atmospheric and climatic conditions as these, and they are the most weighty ones of their kind, an inferior degree of importance only is found by experience to attach.

As to the exciting causes of Typhus, the great, if not the only one, is the specific poison of the disease transmitted from person to person by contagion or fomites. Evidence of propagation of the fever by communication between the sick is seen in its epidemic spread when it enters a community of susceptible persons, and even more conclusively in the way in which persons exposed to none of its predisposing causes catch the fever when they are in close attendance upon cases of Typhus. Nurses in hospitals, where many cases of Typhus are received, invariably get Typhus, no matter under what sanitary conditions they are placed. There appears to be no exception to this rule, unless, indeed, it be that the nurse is personally insusceptible of the disease from a previous attack of it. Medical men and Catholic priests in attendance upon numerous Typhus cases are also almost sure, sooner or later, to get the Fever, and that they do not fall ill with so much certainty or rapidity as the nurses appears due only to their contact with the sick being less constant and intimate. The contagious matter of the disease seems peculiarly capable of destruction when it is diluted with air. Thus tolerably close communication with the body of a Typhus patient appears requisite for the reception of contagion from him. Casual visitors to fever wards very seldom get Typhus, and in private houses of the better class the disease rarely spreads to the attendants. Extension of Typhus from a hospital to the adjacent streets is unknown, even though there should be hundreds of cases congregated within a very short distance of other buildings. In these respects Typhus differs much from small-pox and some other diseases of its class.

There appears reason to believe that Typhus can be communicated apart from actual intercourse with the sick by residence in a house where the fever has recently existed, and by the use of articles of bedding and clothing that have been recently used by Typhus patients. But, as compared with scarlatina, for instance, the degree to which the contagious matter of Typhus can be thus conveyed by fomites is very inconsiderable, and, in fact, it appears to be very seldom thus conveyed if the simplest means of purification by air and water are employed.

Great immunity from an attack of Typhus is obtained by a person who has once suffered under it. Some, but very few, well-authenticated cases of second attack are on record.

There are many instances where Typhus Fever occurs in individuals who cannot be ascertained to have been exposed to any contagion, and where the readiest explanation of the occurrence of the fever is that it has originated *de novo* from the intense operation of its predisposing causes. Cases of this kind, happening in the absence of epidemic influence, and constituting the first instances in a community among whom the disease afterwards spreads by contagion, have been collected by Dr. Murchison, and must be allowed to have weight in favour of the theory advocated by him, that destitution and overcrowding are by themselves capable of generating afresh the contagious matter. If, to this view, the speculative objection be opposed that a specific self-multiplying matter can only be allowed to have a specific origin, the speculative answer may be given that at some time or other there must have been generated a first case of Typhus, and that the same *de novo* production may therefore occur again. Considerations of this kind, on one or the other side of the argument, are of little importance by the side of Dr. Murchison's actual observations. The most serious obstacle to the reception of this theory arises from the analogy of other specific diseases as to the present production of which by contagion, and contagion alone, there is no question. Thus, in many outbreaks of scarlatina and small-pox, the source of infection in the first instance is often as obscure as in the cases of apparently spontaneous Typhus cited by this author, and there are cases even of children's syphilis originating under circumstances that the most experienced investigator has failed to connect with exposure to the poison. Another consideration which weighs somewhat against the belief, that destitution with overcrowding is the condition required and sufficing for the *de novo* production of Typhus, is that outbreaks of Typhus, apparently spontaneous, sometimes occur in persons under excellent hygienic circumstances. Thus in a case that came under the notice of the writer, two boys living in an institution where every advantage of diet and lodgment was afforded (as may be held proved by the fact the fever did not extend) were attacked within a few hours of each other with Typhus, and the most careful inquiry failed to show that either of the boys had had the opportunity of getting the fever by contagion.

SYMPTOMATOLOGY.—The period of *incubation* of Typhus is not satisfactorily determined. It is so rare for persons to fall ill of this fever after a single exposure that opportunities of ascertaining the point in this way do not often occur. And in practice it is also extremely difficult to get, with any accuracy, at the limits of the incubation period from the times of first and last exposure. It is probable that this period is not constant, but that it varies from a few hours to several days.

The *invasion* of Typhus is generally marked by headache, more or less severe, loss of appetite and general malaise. For a day or two, and in the absence of information respecting exposure, there is nothing to distinguish the outset of Typhus from that of any other fever, unless it be the absence of the positive symptoms of other specific illness. It is particularly difficult to separate the invasion of Typhus from an attack of acute dyspepsia. Rigors are of frequent occurrence, but they are not so definite or so severe as in small-pox, or in the pyrexia accompanying internal inflammation. The sense of chilliness commonly complained of along with the early headache may not amount to actual shivering, and it is often wholly absent. In slight attacks, especially in children, it may not be possible to settle the actual time of invasion. On the other hand, in severer cases the disease begins very suddenly with shiver, headache, and it may be vomiting. For three or four days the symptoms of the invasion period get worse, and are accompanied by sleeplessness and general pyrexial symptoms, thirst, heat of skin, pretty complete anorexia, and usually a very peculiar prostration. In a case of Typhus of any gravity the patient gives in to the disease within the first three days, leaving off his work and commonly taking to his bed by that time. Patients with an actual Typhus rash on them, and having been ill five or six days, do indeed sometimes apply as out-patients of dispensaries and hospitals, but this is incomparably of rarer occurrence with Typhus than with typhoid or small-pox patients.

Before proceeding to state the symptoms of a fully-developed case of Typhus, under the heads of the organs and systems affected, mention must be made of *the general appearance* of the patient. This is so peculiar as to constitute to the practised eye a very ready means of diagnosing Typhus, and frequently even an important element in deciding on the nature of a doubtful case. In an average attack the patient lies prostrate on his back with a most weary and dull expression of face, his eyes heavy and with some dusky flush spread uniformly over his cheeks. In the advanced stages of a severe attack, he lies with his eyes shut or half shut, moaning and too prostrate to answer questions, to protrude his tongue, or to move himself in bed; or the mouth is clenched, the tongue and hands tremble, and the muscles are twitching and half rigid. The dryness of the mouth, the sordes on the teeth and lips, the hot dry skin, and the deafness are other symptoms which strike an observer so immediately as to deserve to be included in the physiognomy of the disease.

The maximum *temperature* reached in the course of the disease is rarely less than 104·9 or 105°, and in many cases it reaches 106·5—107°, sometimes even a higher point than this. This high maximum is, as a rule, in favourable cases only attained once or twice throughout the fever, and generally of an evening; the highest morning temperature very rarely exceeds 106°. The temperature begins to rise at the commencement of the disease, and has been observed as high as 103·8—104·9° the first evening; it continues rising till the third day, when it often reaches 105—107°. The maximum is generally attained in the middle of the first week, between the fourth and sixth days and generally on the fourth day, and then a slight though appreciable fall takes place. There is generally a well-marked remission about the seventh day. In Typhus, though less than in other forms of fever, there is an exacerbation in the evening, and the remission about the seventh day is, in some cases, only indicated by the comparative slightness of the evening elevation which then takes place. In the more severe cases there is no trace of remission at this period, but the temperature maintains itself steadily or even rises a little. The absence of this remission marks the case as likely to be a severe one.

In the second week the temperature rises again, but only for a day or two, and rarely reaches the maximum of the first week. The elevation may be from half a degree to two or three degrees, but averages about three quarters of a degree, often lasts but for one evening, sometimes continues longer.

Between the twelfth and fourteenth day there is a remission, in both the mild and severe forms of the disease; even in cases about to prove fatal, and in those other severe cases which showed no decrease of temperature at the end of the first week. But notwithstanding some remission, the temperature in fatal cases often remains high (rarely however above 105°), and shortly before death a very rapid rise occurs, indicating that the fatal termination is approaching, in some cases the temperature being sometimes higher at this time than at any previous date of the disease. In cases of recovery from an average attack, defervescence generally occurs some time between the thirteenth and seventeenth day, and its approach is sometimes announced by a slight exacerbation which renders the subsequent fall more conspicuous. The return to the normal temperature takes place very quickly. In the majority of cases it is completed within twenty-four hours, often in twelve hours; it begins very frequently in the night, and the abrupt manner in which the fever leaves is one of the peculiar features of the disease, the temperature sometimes falling as much as three or four degrees in the course of a night. By the end of the second half of the third week the temperature has usually returned to its natural standard.

The difference between the morning and evening temperature is smaller in the first week and the first half of the second, not amounting to more than one degree or one degree and an eighth; somewhat more considerable from this time to the termination of the disease, averaging about one degree and a half. The evening rise is much less than in cases of typhoid fever, in which disease the great elevation of temperature which precedes the fatal issue of the disease, is not so marked as in Typhus.

Symptoms referrible to the circulatory system.—The pulse of Typhus is always accelerated; in a case of medium severity in an adult being about 120, in slighter cases not exceeding 80 or 90, while in children (by reason of their age), and in more serious attacks in adults, the pulse reaches up to 140—150, even beyond this to a number which cannot be estimated by the finger. From the time of attack on the one hand to that of improvement or death on the other, the pulse seldom fluctuates or falls; it rises steadily up to its maximum in favourable cases, maintains the same moderate excess for several days, and then subsides uniformly and rapidly. In graver cases the pulse continues to rise until the crisis of the disease is reached. A fall in the frequency of the pulse indicates, in the very great majority of instances, the commencement of convalescence, and a subsequent rise signifies the accession of some local complication. It is said that a sudden fall in the pulse, especially when it has been excessively high, occasionally precedes death. Probably this is sometimes the case when there are obvious symptoms of impending dissolution, but diminution in frequency of the pulse has not been observed to precede other fatal symptoms. The difference between the morning and the evening pulse appears to be only a slight exaggeration of that which is observed in health. Change from the lying to the sitting posture increases the frequency, but not to any remarkable degree.

The character of the pulse is peculiar; it often strikes the observer as being very large and strong, but very slight compression is ordinarily sufficient to obliterate it altogether. In other cases it is distinctly feeble and small, and when most rapid is often scarcely perceptible to the finger. Irregularity of rhythm is observed in some severe cases. A dichrotous pulse is occasionally found, but more rarely than in typhoid. On the other hand, instances are sometimes seen when every second beat of the heart only gives a pulse at the wrist. This circumstance is only transient, and must not be confounded with a real reduction in the heart's frequency.

The heart sounds in Typhus may be natural, but in severe cases they are weak and distant, the first sound especially, as has been shown by Stokes, being deficient in tone even to the point of being quite inaudible.

The capillary system exhibits also important changes in Typhus, showing themselves clinically on the conjunctiva and skin among the external parts, and in congestions of various internal organs. The eyes are often bloodshot, and the skin much injected, symptoms more observed in persons below the middle period of life than in old persons whose circulation is inactive. The injection of skin is sometimes carried to such a point, that the finger drawn lightly over the surface causes a white stripe to appear in the course of a few moments. Besides this general injection, a special eruption results from congestion and extravasation of blood in the vessels of the skin.

This eruption, constituting the measley or mulberry rash of Typhus, is present, at some time or other of the disease, in about 95 per cent. of cases, and forms the principal diagnostic evidence of the fever. It has been described by Jenner and most subsequent writers as consisting of two portions, but between the two every intermediate link may be found. The one is a faint, irregular, dusky-red, fine mottling, looking as if it lay some little distance below the surface of the skin, and were seen through a semi-opaque medium. This appearance is well expressed by the name (which is otherwise inexpressive enough) of "subcuticular" mottling. The other part of the eruption is formed by separate spots of small size and purplish colour, scattered over the mottled surface, and looking more or less superficial. These are the "maculæ" of Typhus. They are irregularly roundish in shape, and in colour vary from brightish-red to livid, fading into the colour of the adjacent portion of skin. At their first appearance, they are often a little elevated, and, exceptionally, are found to be as much so as the spots of typhoid. In the course of a day or two they are no longer felt raised above the surface. The mottling often exists without the distinct spots; the spots rarely without a considerable degree of mottling. Usually, the two exist together, but in slighter cases (in children especially) the greater part of the eruption is formed by the general mottling, while in old persons it consists mainly of the distinct maculæ.

As a rule, the eruption of Typhus appears on the fourth or fifth day; it may, however, be met with as early as the third, and rarely is delayed as late as the seventh day from the onset of fever symptoms. It comes first on the backs of the wrists, the borders of the axillæ, and about the epigastrium; in many cases it covers the whole trunk, and frequently also the arms and legs. More rarely it is met with on the face and neck, but in children especially it may be so copious on the face as to resemble measles.

The eruption takes a variable time, under forty-eight hours, for its complete development, and then undergoes certain changes, which, if life is enough prolonged, end in its disappearance; but from the establishment of the eruption in the first week of the disease, no fresh spots are seen. The spots are at first wholly obliterated by pressure with the finger, but after a few days there is commonly some little yellow colour left when the finger is removed, looking as if the colouring matter of the blood had stained the skin at the injected spots, and later in severer cases pressure fails to remove the maculæ to any considerable extent, owing to an actual escape of blood from the vessels. The duration of the eruption varies according to the relative amount of mottling or maculation, and according to the degree to which the spots become ecchymotic. In slight cases with few distinct spots, and occurring among children, the mottling may not last more than two days, and the skin be then left quite clear. In cases of medium severity, the greater part of the mottling disappears within a few days, going first from the face and wrists; the skin of the trunk still showing a crop of the irregular maculæ, half ecchymosed, and, in their later stages, apparently seated at various depths below the surface. In such a case the whole eruption lasts till the twelfth or fourteenth day. But in severer cases, especially when the general eruption is livid and the maculæ irremovable by pressure, the typhus spots persist later than this, and the small ecchymoses may not disappear until the twenty-first day or even later. Desquamation of the cuticle is not observed as a consequence of the eruption.

Another lesion, of rare occurrence, connected with the circulatory system, is local gangrene. This is more commonly observed in winter than in summer. The toes, the legs, and even the nose may suffer. More frequently sloughs on the sacrum and heels are seen as the result of pressure combined with defective circulation. Large, heavy people, much prostrated, who lie helplessly on their backs day after day, suffer most from these accidents.

Other alterations, connected in nature with the circulation, will be considered symptomatically under the headings of the Brain and Lungs.

Symptoms referrible to the Digestive System.—The tongue of Typhus Fever presents every variety of appearance. In the earlier stages it may be unchanged or covered with a thick white fur. Among adults uniformly, as the disease advances, the tongue becomes dry, the fur forming a rough brown coating over a red mucous membrane. Often the tongue is so hard and the whole of the mouth so dry, that from this cause alone there is difficulty in protruding it. At the end of the second week, in favourable cases, the edges get moist, and the tongue cleans, the fur disappearing molecularly or else in patches, leaving the mucous membrane shiny and red. In severer forms of Typhus, with a variable amount of fur, the dry tongue cracks and bleeds, giving rise to black sordes during the disease, and to fissures of which the remains persist after recovery. The tongue may be intensely red and cracked, without there being much fur, and in such cases the characteristic tongue of typhoid is closely simulated. The papillæ are rarely enlarged at any period.

The mucous membrane of the mouth and throat gets, like the tongue, dry and covered with sticky masses of mucus. The lips, in bad cases, become covered with black sordes like the tongue.

Thirst is a symptom met with very uniformly, and from the earliest period of Typhus. It does not give way until, as convalescence begins, the natural secretion of the mouth suffices to keep the surface moist. Extreme loss of appetite is another symptom of equal constancy. For a few days, indeed, in mild cases, the patient can be persuaded to take light food, but as a rule refuses everything solid, retaining a desire for stimulants only. These, too, are soon distasteful, and then the only thing relished is cold water. Vomiting is a less uniform symptom. It sometimes, indeed, occurs at the outset, and occasionally forms a troublesome complication in the progress of the fever.

Diarrhœa, with tympanites, is another digestive symptom of not infrequent occurrence. It appears to have much to do with the regimen under which the patient is put, inasmuch as it certainly occurs more in the practice of some institutions than of others in the same epidemic. It is worth while to insist upon this cause of diarrhœa, since its occurrence at one time, or in one place, might otherwise be mistaken for a special type of the disease there. Thus in the practice of the London Fever Hospital of recent years, diarrhœa has been seen in at least one-third of the cases of Typhus. In the epidemic of 1856, it was practically absent there; at Liverpool, during the present epidemic, there has been little of this complication; and even in the present London epidemic it has been absent in the Typhus treated at some workhouses. The symptom appears to be ascribable in some measure to the greater amount of liquid food that is forced upon the deranged stomach in the practice of some physicians. The plan of the London Fever Hospital is to give at short intervals as much liquid nourishment as the patient can be made to take. If the occurrence of diarrhœa do really result from this circumstance, it must be confessed to constitute a slight drawback to a plan of treatment, which for its aggregate results is not the less to be warmly advocated. When diarrhœa is present there is often considerable tympanites, and some gurgling may be felt, but it is seldom fine or confined to the cæcal region. The abdomen is in such cases slightly tender on pressure, but such tenderness is more about the epigastrium than elsewhere.

On the other hand, in many cases of Typhus the bowels are constipated, but they are readily acted on by purgatives. The stools in this fever differ from those of typhoid, even when diarrhœa is present. They are usually dark, and are of every consistence, but they do not exhibit the appearance of powdery matter suspended in liquid. Their reaction is stated to be acid.

There is an obscure connexion between dysentery and Typhus, the fever appearing under certain circumstances to be generated by persons suffering from dysentery. When this connexion has existed, the tenesmus and frequent bloody stools of dysentery have been observed to complicate cases of Typhus.

The writer has seen peritonitis occurring in one instance only among some thousands of cases of Typhus that have come under his notice; in that case it resulted from the rupture of a multiple abscess in the spleen following on endocarditis.

Symptoms referrible to the respiratory apparatus are so common and so important that they must be regarded as essential parts of the disease. In most cases of Typhus, during the second week there is some little dry rhonchus found at the posterior bases of the lungs. The chest should be examined daily for this condition, even when there is no objective lung symptom. By care in this respect, the accession of the next series of symptoms may constantly be prevented. These consist in increased duskiness of the face, livid flush on the cheeks (not specially on the malars), dusky colour of rash—conditions indicating defective aëration of blood—and these may exist without any cough, but are almost always accompanied by some increase in the frequency of respiration. The patient will make no complaint, but the smallest degree of any of these symptoms should at once point attention especially to the lungs. Often dry rhonchi at the bases will alone be found, but frequently also dulness of one or both bases. About the lower six inches of one base is the amount of dulness usually reached, but it may extend up to the spine of the scapula on each side. With the dulness are found increased vocal fremitus, high-pitched respiration, and at the earlier and later stages of the dulness, coarse muco-crepitation. Fine dry crepitation is less frequently heard over the solidified lung. It cannot be too strongly insisted on, that with all these evidences of lung mischief, there may be no cough, and no expectoration, up to the time that redux crepitation begins, and very likely nothing beyond the duskiness that has been mentioned to call attention to the state of the chest. Upon the occurrence of secondary crepitation, cough often becomes more troublesome, and if there have before been no expectoration it now appears, and consists of a semi-transparent tenacious mucus, scantily aërated, and frequently discoloured with varying blood-tints, as in idiopathic pneumonia.

Bronchitis and consolidation of the lung in Typhus are very apt to improve about the period when the fever itself reaches its turning-point, which has been stated to be usually about the fourteenth day in adult patients with moderately severe Typhus, and these lung states do not often constitute a superadded disease after the end of the third week. But when they are present they constantly obscure the occurrence of favourable crisis, and protract the total convalescence of the patient for several days. The physical signs in cases of recovery usually disappear pretty rapidly, but when dulness has been considerable it may not be quite got rid of for some weeks, although the patient be gaining strength, and have no other evidence of chest mischief beyond this dulness, and a pulse that keeps up above its natural standard. In cases of Typhus fatal from lung complication, the lividity of surface and the physical signs get gradually worse, and generally (but even then not invariably) there is visible embarrassment of respiration.

The nervous symptoms in Typhus are constant and prominent, and it is probably through the nervous system that the poison of the disease primarily operates. It is from certain of the nervous symptoms that the name of Typhus was originally conferred upon continued fever.

From the very outset of the illness these symptoms occur, consisting in rigor, headache, and weariness of body and mind. The amount of head symptoms is greater in proportion to the severity of the attack and the age of the patient. Restlessness and loss of sleep are, even in the slight attacks of children, pretty constant from the first. Sleeplessness often continues up to the time of crisis, and constitutes one of the most distressing parts of the illness to the patient; and even if he gets a good deal of intermitting sleep, he will frequently protest, with many complaints, that he has not slept a wink The headache is often intense, but is a dull and heavy, and not a sharp pain, and is accompanied with some giddiness and with noise in the ears. It gets worse through the first week, and then gradually disappears, rarely lasting longer than the tenth day. Before the cessation of headache, the intellect is heavy, the faculty of appreciating dates and intervals of time being notably confused.

In a large proportion of cases, delirium is a symptom of Typhus. It supervenes usually between the fourth and eighth days, the headache going off as the mind begins to wander. Subjectively, as learnt from the statements of convalescents, the delirium is formed by utter confusion about time and place and people, and even about personal identity. The patient often has fancied that he is two or three people, and is undergoing several sets of miseries and horrors. Objectively delirium is of very various amount, and in character may be active and maniacal, or low and muttering. Much active excitement is rare in Typhus, but extreme degrees of it are occasionally seen, the patient praying, bawling, blaspheming, according to his habitual turn of mind, or leaving his bed to escape from imaginary ill-treatment. Suspiciousness is a not infrequent form of delirium, and the obstinate refusal of food that comes of this mental state may be carried to a degree that itself is fatal to the chance of recovery. Acute delirium commonly passes after some days into the low muttering form, the form which is more usually the character of the delirium from the first. In this the patient lies talking quietly to himself about matters that interested him at the time of his seizure, or on subjects suggested by what is going on, or he supposes to be going on around him. In severer Typhus, the muttering delirium passes into a heavy stupor, and tremulousness of the tongue and hands, with twitching of the muscles (*subsultus tendinum*), is then commonly observed. In very bad cases the patient cannot be roused from his coma, or a few days before his death he falls into the state known as coma-vigil, staring vacantly and with fixed eyes while in a state of complete unconsciousness. To the severe coma, rigidity of the muscles, fumbling at the bed-clothes, and loss of power of swallowing are added before death. Convulsions are another symptom of occasional occurrence, and of almost certainly fatal augury. They are epileptiform in their character, lasting a few minutes only, and giving place to profound coma, in which the patient dies, with or without a repetition of the convulsion. Convulsions in Typhus are almost always associated with albuminous urine, and, in a few cases where this has been investigated, with urea in the blood.

If the head symptoms have been prominent, and yet the patient recover, a childishness of intellect is often left for a few weeks. In very rare cases, the patient has remained insane for some time, but in such of these cases as have come to the writer's knowledge the previous history of the patient's mind was not satisfactorily made out. As a very rare condition indeed, softening of the brain, proving fatal, shortly after the fever has subsided, has been witnessed.

All cerebral symptoms are severer, and the delirium is commonly earlier and more active in persons of the better class of life, when they happen to contract Typhus, probably on account of the habitually greater activity of their brains.

Of the special senses, that of hearing is chiefly affected; besides noises in the head, deafness is of very frequent occurrence, beginning at the end of the first week, being slight, or nearly total in amount, and persisting even after the advent of convalescence. Nothing is to be seen in the ear to account for it. The eyes are suffused, and the conjunctival vessels injected. The sight is rarely affected, but much light increases the headache. The pupils vary a good deal from a condition of medium dilatation to one of great contraction. Their size has not, in the writer's experience, a very constant relation to the mental state of the patient, nor to the general severity of his disease; but small pupils are the rule when coma is present. Some sluggishness of the pupil to the action of light is then also frequently observed.

Kidneys.—The urine of Typhus has not been sufficiently investigated, and many of the following statements rest wholly upon the writer's own observations.

In quantity it directly represents the amount of fluid ingested, regard being had to the other ways in which the body gets rid of water. The quantity has been found greatest in the first week of the fever; about the same or slightly less, in the second; and notably less during the third week after the commencement of convalescence; the mean of several cases and of several days being taken for comparison. A belief exists among good authorities that much ingested water is retained in the body during the whole stage of pyrexia, but of this there appears to the writer to be no evidence that will bear scrutiny. The occurrence of diarrhœa diminishes *pro tanto* the amount of fluid carried off by the kidneys; as to the perspiration, it is not yet demonstrated what alteration in its amount goes along with the increased temperature of the body; it is commonly assumed, and is probable, that very little water is got rid of by the dry-feeling fever skin. An increased amount of urine is said often to accompany the crisis; this has been observed occasionally by the writer, but with no approach to constancy.

The colour of the urine in Typhus is variable. In most cases it is darker than usual up to the turn of the disease; it then becomes natural in colour, and after the third week it is commonly pale. It is very rarely pale throughout. The reaction is acid, probably not more so than in health; and in a case quantitatively examined by Parkes, the free acid reached to only half the normal standard.

The twenty-four hours' urine is often quite free from deposit; but at some period or other of the disease it is usually turbid from lithates. A deposit of lithates has been said to occur as a critical discharge; but in the cases examined by the writer, this was observed quite as often at earlier as at later periods, and most frequently a day or two before convalescence. The daily amount of uric acid at the height of the fever appears (from one analysis by Dr. Parkes, and one by myself) not to be in excess of the healthy quantity. In Parkes' case, the sulphuric acid excreted was rather high; the phosphoric acid has not been estimated.

The chlorides of the urine are greatly reduced during the pyrexial period of Typhus. This partly results from salt not being ingested; but there must be some other cause for their disappearance from the urine, since all chlorides may be withdrawn from a person in health, and yet the urine will continue to contain considerable quantities for some time after. In pretty severe cases of Typhus the abolition of the chlorides may be total; but usually there is a small amount secreted, estimated volumetrically at from two to three grains in the twenty-four hours, and just giving an opalescence when acid nitrate of silver is added to the urine. Before the advent of convalescence, the diet remaining the same, the chlorides reappear in some quantity, undergoing a gradual increase, though the quantity ingested remains the same from day to day. It is not ascertained how far their previous diminution is made up for by greater excretion of them during convalescence. Their disappearance is not connected with diarrhœa, nor is it due to the accession of pneumonia.

When common salt is taken into the stomach as a medicine in the early days of Typhus, while the natural chlorides are being excreted in diminished quantity, it does not pass out by the urine. When taken at a later period, just before convalescence, it is found freely in the urine. In fatal cases, it does not appear up to the time of death. In one such case, where twelve drachms of salt were given to a patient on the ninth and tenth days of Typhus, mere traces of it were excreted by the kidneys, up to the time of death on the seventeenth day; and upon examination of the blood, a certain small excess, but not accounting for one drachm of salt in the whole volume of the blood, was detected. It is probable, therefore, that the chlorine attaches itself to some solid tissue of the body; or that the excess is diffused alike through all the tissues, and that its retention is intimately connected with the febrile condition.

The daily excretion of urea in Typhus, as deduced from sixteen cases accurately observed through the several stages of the disease, is at first considerably above the normal amount. Taking one case with another, the daily quantity during the first week—the patient being fed on low diet, milk and beef-tea—may be stated as about double that of the fourth week, when he is sitting up and eating his fill of meat. The increase is found at the earliest day at which the urine has been examined. In three fatal cases, it did not diminish in quantity during the time that the urine could be procured. In cases of recovery the amount of urea gets gradually less, sometimes, but not always, showing a special decrease about the time of crisis.[1]

The urine of Typhus is occasionally albuminous. In fifteen male cases of various severity, examined throughout their course (some as early as the third or fourth day, and most as late as the fourth week), albumen was found in two cases. One of these was fatal on the tenth day without complication, and the urine contained much albumen on the fourth, fifth, and sixth days, the only occasions when it could be collected. In the other case, a trace was found on the eleventh day, but none before or after; here the patient died on the twenty-fifth day, from one of the rarer sequelæ of the disease. Albumen is only found in Typhus of considerable severity; but in some of the cases examined, though the fever proved fatal, there was none in the urine at any stage. The proportion of cases here stated to have exhibited albumen is below that which is reported as the experience of most observers.

A trace of sugar was observed at one time or another in nine cases out of fourteen, when it was sought for. It appeared at any period between the sixth and twenty-seventh days, and only lasted a day or two. It was probably no more than often occurs in health, and was certainly of no clinical significance.

Convulsions, as connected with kidney disease, have already been considered under brain symptoms. Retention or else involuntary passage of urine is frequent in severe cases.

Generative System.—The catamenia are sometimes present in patients on their admission to the London Fever Hospital; occasionally to a considerable degree. If not present at the earlier stages, the catamenia do not usually appear during the progress of the fever nor during the portion of convalescence that the patient passes in hospital. When pregnant women get Typhus, if they are past their sixth month, they frequently miscarry, but this accident adds very little to the danger of the case, Typhus Fever herein differing much from typhoid or scarlatina.

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[1] It must not be forgotten that some portion of the excess is due to gelatine taken in beef-tea. In one case it was attempted to estimate the influence of this element. For two periods the patient was fed on milk, and no gelatine, and between these periods he was fed upon three pints daily of the strongest beef-tea ("so strong it was quite a jelly when cold"), and nothing else. The mean of the two milk periods was compared with the beef-tea period, and an average daily excess of 11·5 grammes (174 grains) of urea was found in the latter. The writer would also like to see the influence of alcohol excluded in certain observations on the urines of fevers.

DURATION.—The duration of Typhus Fever may be measured by the fading of the eruption, by the fall in the pulse, by the decrease of temperature, and by the general improvement in the aspect and condition of the patient. As a rule, amendment begins in all these ways pretty simultaneously, the fall in pulse and temperature being however the most reliable indication of approaching convalescence. Measured by these tests the duration of the uncomplicated fever varies from twelve to twenty-one days, in mild cases (among children particularly) being sometimes less than twelve days, but only in very rare instances reaching twenty-two or twenty-three days. When the fever is complicated, for instance with erysipelas or parotid swelling, the pulse and temperature may keep up beyond this latter date. In about half the cases, they fall on the thirteenth or the fourteenth day. Except when petechiæ on the skin have been very distinct and irremoveable, the eruption usually fades about the same time, and, with very rare exceptions, is altogether gone by the end of the third week.

Relapse in cases of Typhus, meaning by the word recurrence of the specific disease after apparent convalescence, is of excessively rare occurrence. Once only has it happened to the writer to see a true Typhus rash recur, and this was in the case of a woman who lost the eruption on the fourteenth day, and appeared to be getting rapidly well when, after a few days' ailing, the eruption reappeared copiously on the twenty-fifth day and remained visible for more than a week, the patient passing through a second and very severe attack of the fever.

The duration of fatal cases of Typhus extends from two or three days to twenty-one days, which appears to be the limit of risk in cases of uncomplicated Typhus. In civil practice it is uncommon for the fever to prove fatal before the seventh day; but in certain epidemics that have occurred during warfare, death has frequently occurred at an early period, before the development of the eruption. The ordinary duration of fatal cases is twelve or fourteen days. When parotid swelling or other complication appears as the cause of death, the fatal result may be postponed to the thirtieth day, or even later, but it is not then due to the direct influence of the typhus poison.

TERMINATION AND SEQUELÆ.—The *termination* of Typhus in recovery occurs often with great rapidity; the patient who the day before lay prostrate, stupid, and wandering, with only a slight fall in pulse and fever-heat to hint at the commencement of change, becoming conscious, looking comparatively intelligent, changing his position (though now feeling for the first time his utter weakness), and almost suddenly regaining his appetite. This improvement is more usually spread over two or three days, the lividity of the face gradually getting less, the tongue cleaning, the thirst disappearing, the pulse falling ten or twelve beats a day. The fall of temperature takes place pretty rapidly in almost all cases; and if it be retarded while the patient appears to improve in other respects, the accession of some complication is commonly imminent. Occasionally the pulse does not fall below 90—100 for many days after manifest improvement in other respects. This is commonly due to some thickening of the lung remaining behind, and probably in other cases to the weakness of the heart being extreme. On the other hand, a fall in the pulse to a point much below the healthy standard is not infrequent in the early days of convalescence; the natural frequency being resumed in a short time as the patient gains strength.

From the time when the patient begins to mend, he commonly goes on getting appetite and strength from day to day. Any lung symptoms improve at the same time and rapidly disappear. Emaciation, which is seen towards the end of the acute stage, often continues to increase for some little time after the cessation of the febrile condition, but soon the convalescent regains what he has lost and returns to a state of unimpaired health. Frequently he finds himself after a month or two stronger and better than before his illness.

In cases of Typhus terminating fatally through the intensity of the disease itself, prostration, subsultus, and carphology increase, and low delirium passes into complete coma. The pulse and temperature continue to rise; stools and urine are passed involuntarily; food is often obstinately refused, and there is much difficulty in swallowing for a day or so before death. When there is much bronchitis or congestion of the lung, increased lividity of face with quickened breathing and flapping nostrils are observed, but these conditions are often less noticed through the great prominence of the nervous symptoms.

A patient ill with Typhus does not always convalesce immediately upon the cessation of the specific fever. The *sequelæ* of the fever are indeed few and rare especially when it is compared with typhoid or scarlet fever, and very seldom does it leave behind it any permanent impairment of health. These sequelæ consist either in the persistence of some of the local complications that have been enumerated in the description of the disease, or in the advent about the period of convalescence of certain conditions of an erysipelatous nature.

The complications that occasionally continue are consolidation of the lung, which occasionally goes on to gangrene, but generally mends in the course of a week or two; weakness of the heart, leaving the pulse feeble, with a tendency to palpitation for some short time: bed-sores and gangrene of the toes of course have also to be repaired, or may cause death at a late period; and occasionally a patient dies shortly after the twenty-first day from the kidney disease that has been set up in the course of the fever.

When erysipelatous affections make their appearance, it is usually somewhat late in the fever, convalescence being retarded by their approach. But they may also occur during the earlier stages of the disease; and, although for practical purposes they may be regarded as sequelæ, it is probable they have an intimate connexion in nature with the specific disease. Erysipelas itself, following the usual course of idiopathic erysipelas, occasionally occurs about the time of convalescence. It begins at the root of the nose, the fauces being at the same time red, and may spread over the face and head more or less widely, often causing suppuration in the eyelids. In three cases lately observed at the London Fever Hospital, rapidly developed œdema of the glottis had supervened in patients suffering from erysipelas after Typhus. Swelling of the extremities—sometimes consisting in inflammatory exudation into the cellular tissue, at other times connected with phlebitis—and pyæmic affections of the joints, always ending fatally, are other rare conditions of the same sort.

Of this group of sequelæ, the commonest is swelling, or bubo, of the salivary glands, which are liable to become affected at any period of Typhus, but especially in the third week; at any age, but mostly in the adult. In a few hours a swelling forms that is extremely tender, increases rapidly, and, in the majority of cases, goes on to suppuration. The parotid is the gland most commonly affected, and it is not uncommon for both sides to suffer. Next, but at a long interval in frequency, the submaxillary gland is liable to be affected, while the sublingual gland is very rarely attacked, in the only two cases seen by the writer, becoming involved after others of the salivary glands had swollen. Sometimes, more often in the child than the adult, parotid buboes subside without suppuration, but, usually, they go on increasing; in three or four days fluctuation is detected, and if the swelling be not evacuated artificially, it bursts in the mouth, or the meatus of the ear, or upon the external surface. These swellings appear to occur more in some epidemics than in others, and they add much to the fatality of the disease. If they are at all capable of spreading from one patient to another, it is to a very slight degree that this occurs, at any rate, in hospital practice. Upon examination of all the evidence bearing on their nature, Murchison regards these buboes as forming a connecting link between Typhus and Oriental plague.

Inflammatory swellings and abscesses in other parts of the body are occasionally observed after Typhus. Commonly these accompany Typhus only as they accompany small-pox, or other acute specific diseases, but there have been some epidemics in which the lymphatic glands of the groin and axillæ have been observed to swell, again appearing to show an affinity of the disease with plague.

DIAGNOSIS.—The diseases from which it is most often required to distinguish Typhus, are measles and typhoid fever, pneumonia, and certain brain diseases.

The eruption of Typhus is sometimes, though not commonly, a good deal like that of measles, and it appears about the same day after invasion. If it should happen that a child is attacked, and the source of contagion is unknown, there may be real difficulty in distinguishing the two diseases. Coryza, when present and distinct, points to measles. The eruption of Typhus is of a smaller pattern than in measles, and scarcely ever has any crescentic shape. The occurrence of spots on the face by no means excludes Typhus. Much elevation of the rash is in favour of measles. If the diagnosis have remained difficult up to the sixth day, it may then usually be made with certainty, by noting the sudden fall of temperature that then characterises measles, and that does not occur so early or so completely in Typhus. Still even this means is only available for cases uncomplicated with pneumonia.

From typhoid fever, Typhus is usually pretty easy of diagnosis. Minor elements of distinction are, the nature of the fever prevailing in the same house and neighbourhood, and the comparative immunity of old people from typhoid. In typhoid, the invasion symptoms are far more insidious than in Typhus; rigor and headache are less marked. In the early stages of typhoid, epistaxis is sometimes observed, a symptom very rare in Typhus, unless complicated with scurvy. The eruption of typhoid appears later than that of Typhus, rarely being met with before the seventh day in typhoid, while in Typhus its appearance is very seldom postponed so late as this. The mottled, dull-red eruption of Typhus, with its irregular, non-elevated (at least after their first day), and often persistent spots, is not often closely simulated by the scantier eruption of lenticular, rose-coloured spots of typhoid. But the most essential distinction between the two eruptions is, that that of Typhus comes out in one single crop, while in typhoid fresh sets of spots appear day after day, and each spot lasts only three to four days. Diarrhœa is a much more frequent symptom in typhoid than in Typhus ; but in the particular case, the presence or absence of diarrhœa is not much to be relied on for distinguishing the two fevers. The character of the stools is more distinctive; in Typhus they are natural or dark in colour, and, if loose, of muddy consistence ; while in typhoid they are yellow, and consist of powdery-looking matter suspended in liquid. In typhoid, the stools are alkaline, and contain crystals of triple phosphate ; but the stools of Typhus come to resemble them in these respects when there is diarrhœa. The tongue does not give much help in diagnosis, for, though the typical tongue of Typhus—hard, thick, and with much dry brown fur—resembles little the typical tongue of typhoid, flat, red, dry, and cracked, with little or thin fur ; yet either of these descriptions of tongue, and every variety of them, may occasionally be found in either disease. That the tongue should be persistently moist, is a circumstance pointing much to typhoid. Considerable fluctuation of the pulse, and great evening rise of temperature, are points in which typhoid differs from Typhus, but to which appeal can rarely be wanted to guide diagnosis. The duration of the two fevers will commonly afford a point of distinction, if a case should by possibility have remained doubtful throughout: Typhus does not last more than three weeks, and, in the attacks with obscure rash (where confusion with typhoid is most possible), generally about a fortnight only; while, in typhoid, the fever, as evidenced by the pyrexia and the eruption, goes on to a fourth week, and may go on to the thirtieth day, or even later. In referring to duration, as distinguishing the two diseases, it is, of course, necessary to exclude complications that may be keeping the patient ill, after the specific fever has left him.

With idiopathic pneumonia, it will easily be understood that Typhus may be confused, since a species of pneumonia is one of the commonest conditions complicating Typhus. The compressible pulse, the great prostration, and the brown tongue of Typhus, are simulated by certain forms of pneumonia, in which, moreover, the signs that point to the chest may be no more prominent than they are in the lung-consolidation of Typhus. The presence of a Typhus rash is the essential means of separating the fever from the idiopathic local disease, and, without it, the diagnosis cannot certainly be made.

In the same way, the existence of Typhus eruption is the only way in which cases of this fever, complicated with other local lesions, can be distinguished from those local lesions occurring idiopathically—from uræmia or from erysipelas, for instance.

In many cases of Typhus, especially when occurring among drunkards, the patient, without much apparent prostration, has active, suspecting delirium, there is total sleeplessness, the muscles tremble, and there is a considerable resemblance to delirium tremens. But the moist tongue and skin, and the absence of eruption, usually separate this disease from Typhus, from which it also differs in the manner of its commencement.

We have often to make a diagnosis between Typhus and acute idiopathic or tubercular meningitis. Headache is of a sharper character in meningitis, making the patient cry out with the pain, and it persists after delirium has set in, which it never does in Typhus. Instead of the senses being obtuse, as in Typhus, they are usually painfully acute in meningitis, and the countenance has not the look of intense prostration that it commonly has in Typhus. Unilateral symptoms, such as inequality of the pupils, or ptosis, may be seen in meningitis. And in this diagnosis, again, we are guided by the eruption, if it be present, more surely than by any other consideration.

PATHOLOGY.—It has been stated that Typhus is the result of a specific poison having the power of reproducing itself in a healthy person submitted to its influence in sufficient quantity and for a sufficient time. An account of the pathology of the disease should explain what this poison is, how it enters the body, and is given off to infect other persons, and how it operates upon each organ and system to produce the clinical results that have been described. Our knowledge goes but a very little way towards such an explanation. The views of Virchow, Parkes, and Richardson upon the subject afford the most suggestive data, and may be combined into some such account as the following:—

The Typhus poison is a complex organic substance, probably itself in process of decomposition, and capable of producing certain chemical changes in the albuminous tissues and fluids of the body. Upon these changes the symptoms of the disease depend, and in the course of them a fresh amount of the specific poison is produced. The nature of these chemical changes is not known, but the evidence of their occurrence in the albuminous tissues and fluids comes from the changes observed in the blood and urine, and the alterations seen in the structure of the muscles in fatal cases.

The immediate effect of the chemical change that is brought about by the poison of Typhus appears to consist in an alteration of the osmotic properties of the blood. Through this alteration many of the phenomena of fever are evolved. The interchange of material between the blood and the alimentary canal is interfered with,—an interchange which in health is represented by several pints of fluid daily, and which is of as much consequence to the maintenance of the body as the interchange of gases in the lungs. From this interference arise the dryness of the mucous membrane, the arrested secretion of saliva and gastric fluid, the diminished excretion of chlorine from the body, and probably the febrile phenomena.

Another consequence of the chemical change in the albuminous substances of the body is altered metamorphosis of tissue: firstly, in the way of increase, as we see in the great excretion of urea; and secondly, in point of quality, the alteration of the albuminous substances giving rise to new products in the secretions. In the urine Frerichs shows two abnormal albuminous products. In the skin and mucous membrane the peculiar odour of Typhus, in the absence of chemical proof, serves as evidence of some similar change. And the self-producing poison of Typhus may itself be one of these abnormal elements of secretion.

It has been suggested that ammonia, or a compound related to it, is the actual poison of Typhus. To the writer it appears probable that the matter of this poison is of an organic nature less advanced in decomposition than the stage of ammonia, and that the evidence adduced to support the ammonia theory (even if the fallacy of decomposing matter about the mouth have been sufficiently excluded in the experiments upon which this theory is chiefly based) points rather to the production of ammonia as one of the subsequent changes, excretory in nature, of the altered albuminous compounds.

Morbid Anatomy.—The anatomical changes that can be appreciated in Typhus are few, and the only one that is quite constant is a change in the blood. But changes from the healthy standard are seen in the muscles, in the mucous membranes and glands, in the kidneys, in the lungs, and in the brain, and in other organs.

The body of a Typhus patient is not much emaciated, if the patient die at the time usually fatal, at the end of the second or in the third week. The Typhus maculæ, but not the subcuticular mottling, often persist on the skin after death. Decomposition is generally rapid.

The blood in Typhus is particularly liquid. Drawn from the veins during life it coagulates rapidly but very imperfectly, the coagulum being large, dark, and soft. Under the microscope it is stated that the corpuscles are crenate and misshapen, and do not adhere into rolls, but run into amorphous heaps. After death the same appearances are observed in the blood. It is either not coagulated at all, or forms in the heart and great vessels large, very soft clots, and rapidly becomes putrid. Chemical analysis of the blood is as yet extremely imperfect. The proportion of fibrine is stated to be diminished and that of the red corpuscles to be increased, while urea and ammonia are said to have been found in the blood. Lehmann's account is that the fibrin corpuscles and albumen are all in excess at first, but that the amount of corpuscles diminishes in the later stages, causing the blood to have a lower specific gravity. As to the abnormal elements of the blood and the chlorides in it, no sufficient observations have yet been made.

Morbid changes in the muscles of Typhus have been long observed in the heart's tissue. The organ is soft and flabby, and under the microscope the fibres are seen in a state of fatty degeneration, probably identical with that observed in other striated muscles.

Although softening of the voluntary muscles was long ago pointed out by Laennec, and they are known to be often darker and softer than natural, researches into their pathological changes have not been followed out. The elaborate researches of Zenker on the muscles in typhoid were not extended by him to Typhus Fever; but in cases examined by the writer the characteristic changes which were described by Zenker have been several times observed; the granular and waxen degeneration having been well marked in fibres taken from the rectus abdominis and adductor magnus femoris muscles. When the cases examined had been rapidly fatal, the changes were not seen; but when death had occurred in the third week, or later than this from some complication, the degeneration was well marked. Several instances are on record of hæmorrhages into the voluntary muscles, an occurrence which further points to a muscular change in Typhus similar to that demonstrated by Zenker in typhoid.

The mucous membrane of the stomach is occasionally injected and softened. That of the intestines, particularly of the colon, is not uncommonly inflamed, its vessels being intensely engorged, and soft lymph being sometimes exuded on its surface. This condition is met with when during life there has been much diarrhœa. The agminated glands, and the solitary glands of both large and small intestines, may be found enlarged, especially in children who have them naturally very visible. But under no circumstances is there any deposit in these glands, nor does the ulceration of them which is so constant in typhoid, ever happen in Typhus Fever. In those epidemics where dysentery has complicated the fever, the characteristic lesions of that disease have been observed.

Of the glands, the liver and spleen are frequently hyperæmic, large, and softened. The spleen is sometimes pulpy in consistence and enlarged to twice its natural volume ; this is seen more when the disease has been fatal at a late period. When the salivary glands have been swollen in Typhus, inflammation and softening of the gland tissue itself is the lesion usually observed; and if the disease is advanced, suppuration and sloughing of the gland substance and the interstitial areolar tissue. Some authors have insisted that the pathological changes in these glands begin in the areolar tissue between the lobules, but in the writer's experience this is not the rule. Under the microscope abundance of oil-globules and granules, and of pus cells are found, and the gland cells are full of oil-globules. The pancreas is frequently injected, but is not known to suffer any change resembling that of the salivary glands.

The kidneys are not much or often affected by Typhus. Their commonest deviation from health is congestion, the organs being large and somewhat friable. In the rare cases that prove fatal by convulsions, the kidneys are either found the seat of old disease, or they are in some stage of recent engorgement up to actual acute nephritis, or there may be no lesion whatever detected in them.

The bronchitis that is so very common a condition in fatal Typhus offers no appearances after death requiring special comment. The consolidation of the lungs, also met with in most fatal cases, consists either of true pneumonia or (more usually) of hypostatic congestion. In the latter the posterior parts of the lung are dark, non-aërated, friable, with a section that is not granular, as in pneumonia, and from which much dark serosity exudes.

The nervous system commonly shows after death no lesion whatever to account even for intense head symptoms. The utmost change that is usually observed is some fulness of the sinuses, coarse injection of the meninges, and increased vascularity of the brain substance, and none of these conditions reach any intense degree. Occasionally a film of hæmorrhage is seen in the cavity of the arachnoid, and the amount of serum in the sulci and ventricles is greater than usual, but neither of these conditions appears to have any connexion with the brain symptoms during life. Actual meningitis has, however, been observed in Typhus, in such a way as suggests its occurrence more in some epidemics than in others. It is described by the physicians of the London Fever Hospital in their works, published in 1830, as of no infrequent occurrence; but in the course of the last ten years, meningitis has certainly been very seldom seen there as a post-mortem appearance in Typhus. But in the last few years, a form of fever, akin to Typhus, if not actually identical with it, has been observed in America to be often complicated with meningitis of the brain and spinal cord. And in the present year (1865) a Russian physician, Dr. Kremiansky, describes a hæmorrhagic inflammation of the dura mater as of frequent occurrence in persons dying of Typhus in St. Petersburg.

PROGNOSIS AND MORTALITY.—In Typhus, these are affected by nothing so much as by age; but to some degree by temperament and habits, by social position and nature of previous occupation, and also by the characters of the prevailing epidemic.

The rate of mortality from Typhus in a community attacked by it is usually stated much too high, the experience of hospitals, into which few children are received, being taken as the basis of calculation. When every attack, in persons of all ages, is included, the mortality of Typhus is about ten per cent. But when such cases only as are ordinarily sent to hospital are considered, the mortality is about twenty per cent. When age is compared strictly with age, however, this difference, either wholly or for the most part, disappears. Between one hospital and another, or between hospitals and cases treated at their homes, there are indeed some apparent differences of mortality, but the causes of such difference (when real and not dependent merely upon age or accident) are not of a nature that any general statement of them can be made.

The death-rate of children under ten years of age, attacked by Typhus, is about 5 per cent.; that of persons over sixty years is 66 per cent. or upwards, of those seized by the disease. Between the two there is a regular gradation of fatality. For example, at Greenock, the death-rate, at seven periods of life, was recently found to be, under ten years, 5 per cent.; between ten and twenty, 8·6 per cent.; between twenty and thirty, 15·6 per cent.; between thirty and forty, 21·5 per cent.; between forty and fifty, 42 per cent.; and over fifty years, 66·6 per cent.; the mortality increasing with each decade of age that the patient had reached. The death-rates of the London Fever Hospital, analysed in detail by Murchison, gave figures corresponding in the main with these, but all of them slightly higher through the severity of the cases that are presented to that institution. The difference in mortality, according to age, is so great and so universal, that the caution may well be given that no comparison between different methods of managing Typhus can have the slightest value which does not accurately allow for this overshadowing influence.

Bulky, lymphatic, and fat people are more likely to die than those of a different conformation when they are attacked with Typhus. Negroes, treated in the London Fever Hospital, have been observed to have the fever more severely than whites. People of a better class of life, though seldomer attacked, are believed to experience a larger mortality than the poor. Habits of intemperance very seriously add to the unfavourable prognosis.

Occupational differences only affect the prognosis of Typhus in so far as they have involved extreme exhaustion and fatigue, persons who are attacked under circumstances of that kind usually having a high mortality. Overworked soldiers, doctors and nurses, for example, get the disease with peculiar severity. And if the patient try to keep about, going on with his work until he takes to his bed from sheer inability to stand, he materially diminishes his chance of recovery.

The conditions occurring in an attack of Typhus, which give especial gravity of prognosis, are as follow:—Very abundant rash, with spots scarcely affected by pressure; considerable duskiness of surface; a high maximum temperature, as 107° or above; continuous rise of temperature up to the end of the first week; a sudden great rise of temperature in the third week, this being, it is stated, of fatal significance; very weak pulse, with inaudible first sound of the heart; very rapid pulse, death being almost certain if in the adult the pulse exceed 150; lung complications of all kinds make the prognosis bad according to their amount; early delirium; severe and active delirium, with complete sleeplessness; profound coma, and especially coma vigil; intense prostration and subsultus; convulsions, which are almost certainly fatal; albumen in the urine; obstinate refusal of food; vomiting; uncontrollable diarrhœa. Any of the erysipelatous conditions noted as occurring towards the end of the disease contribute seriously to reduce the patient's chances of recovery.

THERAPEUTICS.—Typhus Fever, like other diseases of its class, cannot be cured nor its duration shortened by any means at present known to medical science. Its symptoms may be combated, and its complications may be treated, while the patient's strength is supported through the time of the fever, but we know of no way of encountering the specific disease. Upon a full recognition of this truth, the treatment of a case of Typhus will be most satisfactorily based. If we propose to ourselves to give the patient the best possible opportunities of recovery, our treatment will be more successful, than if we direct our efforts to cutting the disease short by any supposed methods of cure.

In a disease which lasts two or three weeks, in which the metamorphosis of tissue is increased, and in which ordinary food cannot be taken, the patient must be kept up by nourishment appropriate to his new condition, or he will die, as a healthy person deprived of food for the same length of time would die. The essential part of the management of Typhus consists in giving this appropriate nourishment, and in preventing the patient dying from the want of it while the curative processes of nature are going on.

The character of the nourishment to be given requires some detailed consideration. In the early stages of the fever, if the patient have appetite, he may be allowed anything not positively noxious that he has a fancy for. As his dislike for food increases, he will still consent to take liquids and sick-room delicacies. But soon there comes a time, in every severe case of fever, when everything but cold water is distasteful, and when food has to be administered like so much medicine to the unwilling patient. At this time, the digestive functions are in more or less complete abeyance, and the nutriment given must be such as requires the simplest processes for its assimilation. Foremost among nutriments of this kind, experience has put beef-tea and broths, milk, eggs, and alcoholic drinks.[1] Bread, arrowroot, jellies, are other suitable articles; and the nurse who can manage a good deal of variety in the choice and combination of such things as these, does much for her patient's chance of recovery. Samples of such variety are Gillon's meat-juice; vermicelli in beef-tea; chicken or veal broth; mutton broth with rice or bits of toast; eggs in custards or beaten up with milk or with wine; blancmange of isinglass or ground rice; syllabubs or wine-whey; barley-water or thin arrowroot with milk; weak tea or coffee with milk.

For drinks, lemonade, soda-water, currant-water, cold weak tea without sugar or milk, or any of these iced, may be allowed at the patient's choice. Often food is taken cold when hot is refused. But even of this light diet a little only can be taken at once, and it therefore becomes desirable that it should be given frequently. Every two or three hours the patient should be fed, and if he be in a drowsy state he should even be roused up to take his nourishment.

But of these means of giving support, there is none more important than alcoholic drinks judiciously used. It is not every patient that requires alcohol; children rarely do, and about half the adult cases admitted into hospital may be treated without any. But it is especially in two classes of patients that we need to give stimulants: those who cannot take a sufficient quantity of other kinds of nourishment, and those who are in health habituated to the use of strong drinks. Besides the use of alcohol as an aliment, it has also a medicinal effect upon the nervous and circulatory systems, and its full employment will much depend upon whether this effect is desirable. The cases in which alcoholic stimulants are most serviceable are (1), in old people almost universally; (2), cases of great prostration, with low delirium and coma; (3), cases where the pulse is very compressible, and the first sound of the heart feeble, also when the pulse intermits and usually when it exceeds 120 in frequency; (4), cases where the extremities are cold and the surface is livid; (5), where there is much congestion of the lungs; (6), where there is any erysipelatous complication. In a great many cases of Typhus alcohol is unnecessary, and appears to do actual harm when there is violent maniacal excitement at an early stage, and also with young people in whom, without notable depression, there is much bronchitis, or in whom true pneumonia can be diagnosed. Alcohol is rarely wanted before the appearance of the eruption, and is most needful in the second or third week as the patient is approaching the crisis of his disease. For ordinary cases requiring alcohol, the strong wines are best adapted, while lighter wines with water form excellent drinks. Beer is a very good form of giving alcohol with other nourishing principles, and it is often craved for by the patient. Severe cases, particularly in old persons and in drunkards, require spirits, which may be given mixed with beef-tea, with milk, or with eggs. A moderate allowance to an adult, suffering under pretty severe Typhus, with dry tongue, moderate delirium, and weak pulse of 120, would be a bottle of claret or half a bottle of sherry daily. A bad case, with livid features, tremulous muscles, much low delirium, with coma, and a very weak pulse of 140 or 150, may often have 12—20 ounces of brandy or whisky daily distributed in hourly doses. It often happens that in such cases a patient for whom there might otherwise be a chance, obstinately clenches his jaws against his nourishment, or is made sick by it; it is then sometimes possible to tide him over the time of crisis by frequent enemata of beef-tea and brandy, which are usually well retained, even if there have been some tendency to diarrhœa.

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[1] The writer has no intention to side in the controversy concerning the food character of alcohol. He accepts the evidence that much ingested alcohol is got rid of by the excretory organs, or is retained for some time in the tissues after the manner of many medicines. But with food in its widest sense, as what keeps up the vital functions, the physician will have little hesitation in classing alcohol, who has observed the common case of an habitual tippler maintaining for years a fair standard of bodily health upon a quantity of other nutriment wholly insufficient by itself to maintain such health. And to such a case a fever patient offers some resemblance. He, too, may not be able to take enough of other food to maintain him, but alcoholic drinks will help him not to starve. And thus the writer judges them to have a food value apart from their medicinal action.

But though what has been said is, in the writer's experience, the essential part of the treatment of fever, there are many symptoms and complications that can be met by medicinal agents. The thirst is best relieved by acidulous draughts, and there is no better medicine for slight cases of fever than twenty drops of dilute hydrochloric acid in an ounce of water. The headache, sleeplessness, and delirium are very frequently lessened, even though there should be a good deal of suffusion of the eyes, by opium; the writer has constantly given five minims of laudanum every four hours, or else a night dose of fifteen minims, with considerable advantage to these symptoms. He has avoided opium when the pupils are very small, when there is coma, and when there are serious lung complications; but with these exceptions he finds no contra-indications to the use of the drug. If opium be given with the object of soothing violent delirium, it should be in full doses at night, and not in small frequent doses: but if possible it is better not then to use opium. Combined with a small quantity of tartar emetic, opium has an increased power of relieving headache and of inducing sleep. When it is desired to use a sedative, but to avoid opium, good results have often followed from a grain or more of extract of cannabis indica given at night. Strong coffee is said to have relieved headache; the writer has seen no effect from it, either upon headache or coma, in the few cases where he has employed it. Cold lotions to the shaven head and blisters to the forehead are each of use in many cases of severe headache, and they are means that are especially applicable in the cases just mentioned where opium must not be used. In early furious delirium, two or three ounces of blood have been taken from the temples with good results; doubtless if meningitis were diagnosed, this would be right practice then also. When the patient's delirium causes him to leave the bed, there is no means of restraining him equal to the care of an experienced nurse, but there is no objection to mechanical restraint, for if it be effectually applied the patient often ceases to struggle and so saves his strength. From deep coma patients may sometimes be roused by blisters to the forehead and nape or to the shaven scalp. Subsultus and tremors are said to be peculiarly controlled by camphor and musk. Among stimulating remedies that may be used along with much alcohol in cases of great prostration, spirit of chloroform and turpentine are the most valuable.

The bowels had best be kept open once a day, but slightly confined rather than purged. Any but very gentle laxatives are apt to cause weakening diarrhœa. Should this from any cause be present, draughts of sulphuric acid or of chalk and catechu may be given; and when diarrhœa is severe or obstinate, acetate of lead in draught, or sulphate of copper in pill, are most useful, along with small doses of opium if there be no reason against it. Starch and opium (fifteen minims of laudanum) injections are also of great use. Vomiting is best checked by ice, lime water, or soda water, and by bismuth; sometimes by a sinapism to the epigastrium; if it persist in spite of these remedies, it is well to let the stomach have complete rest for a while, supporting the patient by nutritive injections.

For lung complications of all kinds, one of our most valuable remedies in Typhus is carbonate of ammonia. The bicarbonate (formed by exposing powdered sesquicarbonate to the air till its pungency is gone) is less irritating to the parched mouth, and can be given in larger doses than the monocarbonate. Senega assists the expectorant action of ammonia, and may be given, except for its nastiness, in all cases complicated with bronchitis or with consolidation of the lungs. Another extremely useful remedy for congestion of the lungs, especially in old people, is turpentine, fifteen drops in mucilage. And with these internal remedies it is always right to use counter-irritants; mustard poultices, often repeated, to the back and sides of the chest, being the best form of them.

As for the urinary organs, it is important to be on the watch against retention of urine, and to relieve the bladder duly by catheter. Slight albuminuria itself calls for no special treatment, beyond contra-indicating opium, and being, it is thought by some, a reason for not giving alcohol very freely. When there is much albumen in the urine, or when convulsions have occurred, it is right to give gentle saline purgatives that may act also as diuretics, to use mustard or dry cups to the loins, and to get the skin to act by means of the hot-air bath.

Where swelling of the salivary glands occurs, the chance of their resolving without suppuration has seemed to be increased by blisters over them at an early stage. Cotton wool may be applied over the swelling, and when the formation of pus cannot be avoided the abscess should be poulticed continuously and opened as early as ever fluctuation can be detected in it. It is of great consequence to give extra food to the patient as soon as a salivary gland is observed to swell. Eggs, fish panada, and more stimulants should be given without any limit but his ability to swallow them. Erysipelas supervening on Typhus is best treated by extra food and stimulants, by the tincture of iron internally, and by wrapping the affected parts in cotton wool.

In the management of a case of Typhus it is of great consequence to place the patient in the best hygienic conditions, and if he has been attacked in the close crowded rooms where the disease mostly occurs, his removal to a properly constructed hospital should be insisted on. An ample supply of fresh air of even temperature, of clean linen, of soft but cool bedding, and the services of an experienced nurse, are parts of the treatment as essential as the prescriptions of the doctor. Of hygienic treatment, quiet, cleanliness, frequent sponging, and occasional changes of posture to avoid lung congestion and bed-sores, are the most worthy of mention.

As soon as a patient passes the crisis of the fever, and regains his appetite, he may be allowed any article of food that is good for him in health, and may eat and drink liberally. It often happens that with a tongue still dry and brown, and only just moistening at the edges, the patient asks for meat, and if it is given him finds no ill effects from it. But as a rule it is certainly safer to keep him on light puddings and fish until his tongue has got pretty clean and moist. Beer is generally relished in convalescence more than wine, of which the palate is tired, and good ale or stout may be substituted for a great part or for the whole of other stimulants. The amount of stimulants must be brought to the standard required in health by degrees, and not suddenly. When during convalescence the patient remains childish in intellect, or wandering in his mind, it is a reason for giving plenty of food and wine, not for diminishing his allowance.

Of the prophylactic measures to be used against Typhus little need be said, as they consist almost entirely in an avoidance of the predisposing causes that have been enumerated. Persons in attendance on the sick should not be overworked, or deprived too much of their natural rest and exercise, and they may be further guarded against the reception of the fever by the use of disinfectants, of which fresh air and cleanliness are incomparably the most important. Lime whiting and repapering (after lime whiting) of infected rooms, stoving the bed and bedding, boiling the patient's linen, or soaking it in water impregnated with chloride of lime, and the use of this substance or carbolic acid in the water employed for sponging his body, are other means that should be employed for avoiding contagion.

VARIETIES.—The varieties of Typhus are few, and consist chiefly in different degrees of severity. One epidemic may differ from another in its liability to special complications, to dysentery, or to cerebral inflammation, for example; or in intensity, in this respect being affected by the average age of the community in which it occurs, and by the degree to which the predisposing causes of the disease are in operation. But of such varieties no further consideration is here required.

RELAPSING FEVER.

By J. Warburton Begbie, M.D.

This, its familiar name, has been applied to one of the forms of continued fever, on account of its most characteristic and peculiar feature.

The disease may be defined as follows :—

Definition.—A contagious disease, rarely appearing, except as an epidemic ; marked by its sudden invasion, the pyrectic symptoms continuing till about the fifth or seventh day, when, after the occurrence of a critical evacuation, their abrupt cessation occurs. There succeeds an interval of complete freedom from fever, followed by sudden relapse on the fourteenth day from the commencement of the original attack. The condition of pyrexia is again terminated by a crisis on or about the third day of the relapse, and for the most part convalescence ensues. Not very infrequently a second, with increasing rarity a third, fourth, and even a fifth relapse, has been noticed.

History, Nomenclature, and Bibliography.—In 1843 an epidemic of fever appeared in Edinburgh, Glasgow, and other of the larger towns of Scotland, which, although at first believed to present characters previously undescribed, was soon recognised as being similar to the fever which had prevailed in the former city during the years 1817-18, and likewise to the fever which during these years and the one subsequent, 1819, as well as many previous years, had occurred in Ireland. Carefully observed in 1843, and very ably described by several Scotch physicians, this same fever during a later, though by no means so extensive prevalence, in 1847–48, attracted the attention of other accurate observers, both in Scotland and England. Since the disappearance of the last-named epidemic the disease has been very little seen. In 1851 Dr. Murchison informs us that in London, as well as in Glasgow, there was a considerable increase of Relapsing Fever ; but since 1855, this excellent writer on fever remarks, he has reason to believe that not a single case of Relapsing Fever has been observed in either of these cities.[1] As regards Edinburgh, a very competent authority, Dr. W. T. Gairdner, has stated that he has not seen a single case distinctly referrible to this type since 1855.[1] The writer is able to offer an abundant confirmation of the latter statement, for, during his ten years' service as physician in the Royal Infirmary, dating from May 1855, while having at all times charge of fever-patients, he has never once encountered a case bearing any resemblance whatever to the Relapsing Fever.

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[1] A Treatise on the Continued Fevers of Great Britain, by Charles Murchison, M.D. London, 1862. P. 298.

Several of the physicians who have enjoyed the most extensive opportunities of observing the more recent epidemics of Relapsing Fever, have occupied themselves with an inquiry into its history; it may therefore be expedient here, before entering on the consideration of the phenomena presented by the disease, to make a few observations on the former subject, while indicating at the same time the different names by which it has been described, as well as the sources from which the most reliable information regarding Relapsing Fever is to be drawn. In one of the important discussions which took place in the Medico-Chirurgical Society of Edinburgh during the prevalence of fever in 1844, the late Dr. Robert Spittal called attention to the interesting fact "that the present epidemic seems to be exactly the same in all its important features as an epidemic described by Hippocrates, as having occurred in the island of Thasus, off the coast of Thrace." The chief features of resemblance between the ancient and the modern epidemics are the invariable occurrence of relapses, the marked character of the crisis, and the frequent association with the more ordinary events in the disease, of copious perspirations,[2] hæmorrhages, particularly epistaxis,[a] jaundice,[b] splenic enlargements,[c] and in women the tendency to miscarry.[d] A simple mention of the occurrence of one or more relapses in the progress of continued fever has been made by many writers in their descriptions of different epidemics. This circumstance alone, it is scarcely necessary to observe, does not admit of such cases, which were merely exceptional in these visitations of fever—being considered examples of the form of fever now under consideration. Thus, Dr. Srother, in his account of a fatal fever which prevailed for two years in London, mentions the occurrence of relapses as frequent. "Perhaps," he remarks, "we may find reason to lay some blame on the air for the frequent relapses."[3] Dr. Lind,[4] also, in treating of the contagious Typhus of the fleet, alludes to the same. "Many," he says, "relapsed." Joseph Frank, in his learned account of adynamic fevers, refers to the occurrence of relapses, but that these were not often witnessed, may be gathered from his words, "raro recidiva morbi timenda."[1] These quotations will suffice to establish, firstly, the circumstance that occasional relapses in cases of continued fever had long been observed and described; and secondly, that the fevers thus indicated were certainly not the disease we are now discussing, for in it relapses are not merely occasional, nor even frequent, but invariable. We come then to the well-known work of Dr. Rutty,[2] and in it there is afforded ample proof of the existence of Relapsing Fever in an epidemic form in Dublin, ten years after the London epidemic described by Srother. The former writer, in giving an account of the summer of 1739, remarks, "The latter part of July, and the months of August, September, and October, were infected with a fever, which was very frequent during this period, not unlike that of the autumn of the preceding year, with which compare also the years 1741, 1745, and 1748. It was attended with an intense pain in the head. It terminated sometimes in four, for the most part in five or six days, sometimes in nine, and commonly in a critical sweat; it was far from being mortal. I was assured of seventy of the poorer sort, at the same time in this fever, abandoned to the use of whey, and God's good providence, who all recovered. The crisis, however, was very imperfect, for they were subject to relapses, even sometimes to the third time; nor did their urine come to a complete separation. Divers of them, as their fever declined, had a paroxysm in the evening, and in some there succeeded pains in the limbs." Again, in describing the summer of 1741, the same writer, Rutty, observes: "It seems also not unworthy of notice, that through the three summer months, there was frequent here and there a fever, altogether without the malignity attending the former (an adynamic fever which the author had already described), of six or seven days' duration, terminating in a critical sweat, as did the other also frequently; but in this the patients were subject to a relapse, even to a third or fourth time, and yet recovered." Huxham, in bearing a strong testimony to the value of bark in the advanced stage of "the slow nervous fevers," speaks of fevers which "are frequently attended with dangerous relapses."[3] But neither in the work from which this statement is quoted, nor in his other treatise on epidemics, referred to below, is there any account of Relapsing Fever. Dr. John Clark, in 1777, observed at Newcastle a fever, "the duration of which was uncertain, and some relapsed into the fever." This disease about the eighth or tenth day presented a white or red miliary eruption, and sometimes a more universal red rash, resembling the measles; clearly it, too, was other than Relapsing Fever.[1] Drs. Barker and Cheyne, in the concluding paragraph of the first chapter of their interesting work on Epidemic Fevers of Ireland, indicate the occurrence in the very earliest years of the present century of a fever closely resembling, if not identical with, Relapsing Fever. "Certain it is," these authors remark, "that the fever of 1800 and 1801 very generally terminated on the fifth or seventh day by perspiration; that the disease was then very liable to recur; that the poor were the chief sufferers by it; and that it was much more fatal amongst the middling and upper classes in proportion to the number attacked."[2] The widespread epidemic fever of 1817, and the two succeeding years, was in all probability largely composed of Relapsing Fever. Our chief knowledge of the fever as it then occurred, is gathered from the works of Barker and Cheyne, Harty, Bateman, and Welsh. The progress and distribution of the epidemic has been ably sketched by Dr. Murchison,[3] who has in all probability correctly inferred that the proportion of typhus to the relapsing cases was greater towards the close of the epidemic, from the circumstance that the rate of mortality increased at many places with the advance of the disease. Any accurate account of the distinctions between the two forms of fever composing the epidemic in question is not however to be expected, and indeed cannot be found. Dr. Christison, to whom, as Mr. R. Christison, Dr. Welsh in the preface to his work[4] refers, candidly admitted in 1844 that "at the time when he had observed the same fever twenty years ago, it was the general impression that it could produce common typhus, and *vice versâ.*" At the same time, Dr. Christison remarked that the Fever of 1843–44 was not a new one; it had been described by himself as "Synocha" in his article on fever in Dr. Tweedie's Library of Medicine.[5] Allusion has already been made to the circumstance of the fever of 1843-44 being at the first regarded as a new disease. The statement of Dr. Alison on this point is quite distinct. Writing even in 1847, that able and excellent man remarked, "The epidemic fever of 1843 is now generally admitted to have been a new pestilence, hardly anywhere seen in England, and not known in Scotland before that year, extending rapidly and generally in Scotland, but fortunately causing in itself no great mortality."[1] But although no correct distinction was drawn between the two forms of continued fever which undoubtedly composed the epidemic of 1817, 1818, and 1819, it was otherwise during the succeeding epidemic of 1826. Dr. O'Brien in particular, who published an account of the epidemic as witnessed in Dublin in 1826-27, wrote: "At the commencement of the epidemic two species of fever were distinguishable in the wards of this hospital, which, to use the words of Sydenham, we shall call the fever of the old and the fever of the new constitution. The first was the ordinary typhus of this country, marked by its usual protracted periods, running on to the eleventh, fourteenth, seventeenth, or twenty-first days. This species of fever was far inferior in numerical amount to the other, but far more fatal. . . . The other species of fever, or that of the new constitution, which constituted the bulk of this epidemic, was one of short periods, terminating in three, five, seven, or nine days, but the second of these periods was the most frequent. . . . The patient was destined, perhaps, to be harassed by one, two, or three relapses, which prolonged the whole duration of his illness beyond that of the most protracted typhus—in fact, the liability to frequent relapses was one of the most striking characteristics by which this fever was distinguished from all previous epidemics, at least which happened in our time."[2] In respect to the statement of O'Brien on the mortality of the epidemic at different stages of its progress, Dr. Murchison, by a reference to statistical facts, has ably shown that in the latter part of its continuance the cases of Relapsing Fever had in all likelihood greatly diminished. This result is further corroborated by the statement of Dr. Alison regarding the Edinburgh fever of the years 1826-27; the mortality in the latter year, he has stated, exceeded that of the former.

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[1] Clinical Medicine, by W. T. Gairdner. Edinburgh, 1862. P. 158.

[2] The London and Edinburgh Monthly Journal of Medical Science, vol. iv. for 1844, p. 177.

Καὶ ἐφίδρουν. [a] Ἔστι δ' ᾗσι ἐκ ῥινῶν ᾑμορράγησε. [b] Ἔστι δ' οἷσι ἴχτεροι ἐχταίοισι. [c] Αὐτίκα δὲ σπλὴν ἐπήρθη.

[d] Ἧσι δὲ ξυνεχύρησε ἐν γαστρὶ ἐχούσῃσι νοσῆσαι, πᾶσαι ἀπέφθειραν, ἃς καὶ ἐγὼ οἶδα.—Epidemiorum Hippocratis Liber Primus, Sectio Secunda, Status Tertius.

[3] Practical Observations on the Epidemical Fever which hath reigned so violently for these Two Years past, and still rages at this present time, by Edward Srother, M.D. London, 1792. P. 121.

[4] An Essay on the most effectual Means of preserving the Health of Seamen in the Royal Navy, and a Dissertation on Fevers and Infection, by James Lind, M.D. London, 1779. P. 190.

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[1] Praxeos Medicæ Universæ Præcepta, Auctore Josepho Frank. De Febribus Typhodibus, Partis Primæ, Volumen Primum. P. 214.

[2] A Chronological History of the Weather and Seasons, and of the prevailing Diseases in Dublin, by John Rutty, M.D. London, 1770. Pp. 75, 90.

[3] An Essay on Fevers and their various Kinds, as depending on different Constitutions of the Blood; with Dissertations on Slow Nervous Fevers, &c. by John Huxham, M.D. London, 1750. P. 87.

For a further notice of Epidemic Fevers, see the same author's work, entitled "Observationes de Ære et Morbis Epidemicis ab Anno 1728 ad Finem Anni 1737. Plymuthi factæ." London, 1752.

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[1] Observations on Fevers, especially those of the Continued Type, &c. by John Clark, M.D. one of the Physicians to the Newcastle Dispensary. London, 1780. Pp. 131, 132.

[2] An Account of the Rise, Progress, and Decline of the Fever lately epidemical in Ireland, together with Communications from Physicians in the Provinces, and various Official Documents, by F. Barker, M.D. and J. Cheyne, M.D. In 2 vols. London and Dublin, 1821. Vol. i. p. 20.

[3] Loc. cit. p. 36.

[4] A Practical Treatise on the Efficacy of Blood-letting in the Epidemic Fever of Edinburgh, illustrated by numerous Cases and Tables, extracted from the Journals of the Queensberry House Fever Hospital, by Benjamin Welsh, M.D. Edinburgh, 1819.

[5] Discussion in Medico-Chirurgical Society of Edinburgh, January 3, 1844. Monthly Journal of Medical Science, p. 177.

The epidemic of 1826-27 ended, Relapsing Fever was probably absent from Great Britain till the year 1843, or the very close of the preceding year. At that time there appeared in Scotland an epidemic, of which excellent accounts were published by Drs. Alison, Craigie, Halliday Douglas, and Henderson; while the fulness and accuracy of the treatises written about the same time by Drs. Cormack[3] and Wardell[4] justly entitle them to be styled admirable histories of that epidemic. Three complete years separated the epidemic just referred to and its successor; although, during that period, embracing the years 1844-46, "a few cases of Relapsing Fever continued to be observed, both in

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[1] Observations on the Famine of 1846–47, in the Highlands of Scotland, and in Ireland, by William Pulteney Alison, M.D. Edinburgh, 1847. P. 9.

[2] Medical Report of the House of Recovery and Fever Hospital, Cork Street, Dublin, for the Year ending 4th of January, 1827, by John O'Brien, M.D. in Transactions of the Association of Fellows and Licentiates of the King's and Queen's College of Physicians in Ireland. Dublin, 1828. Vol. v. pp. 526, 529.

[3] Natural History, Pathology, and Treatment of the Epidemic Fever at present prevailing in Edinburgh and other Towns, &c. London, 1843.

[4] The Scotch Epidemic Fever of 1843–44. London Medical Gazette, 1846–47.

Ireland and Britain."[1] In 1847-48, and this is the last occasion in these Islands, Relapsing Fever was again very prevalent, the particular epidemic being, as aforetime, constituted partly of typhus and partly of the shorter fever—cases of the latter, about the commencement of the unusual prevalence of fever, being in considerably greater proportion than those of the typhus. This epidemic of fever prevailed in England, as well as in Ireland and Scotland. In the former, it is probable that throughout its entire progress, cases of typhus were greatly more frequent than in Ireland, and decidedly more so than in Scotland. A very careful observer and instructive writer on the Irish fever has indeed stated, that "cases of genuine typhus were, through the whole epidemic, very rare. Occasional cases did occur, and these became more numerous with the advance of the epidemic." It is right to note here that Dr. Henry Kennedy's opinion, just quoted, is not acquiesced in by all authorities; for example, Dr. Lyons, in his valuable work on fever, makes the following observation:—"While I admit the frequent occurrence of Relapsing Fever in Ireland, I must be allowed to record here my protest against the statements recently circulated on very insufficient data, that Relapsing Fever constituted the large majority of the cases of the famine fevers of Ireland. The contrary of this I believe to be the case; and, having had large and extended experience in the last great famine visitations of Ireland, 1846, 1847, 1848, I can certify that the maculated typhus was the disease which chiefly prevailed; while the Relapsing Fever presented itself only at the close of the great typhus visitation."[2] Of the epidemic fever of 1847-48, many valuable accounts have been written, including those of Dr. Robert Paterson,[3] of Leith, and Dr. William Robertson.[4]

The works and papers which have been already referred to, are among those which contain the most satisfactory information regarding the Relapsing or Short Fever.[5] Other sources will be indicated in the sequel; it may, however, be stated, once for all, that in the exhaustive treatise of Dr. Murchison, the fullest, as well as the most reliable information respecting this and the other forms of continued fever which occur in Great Britain is to be found. This division of our subject may be closed with a notice of the different names under which the Relapsing Fever has been described.

The *Synonyms* are, indeed, numerous, and have been suggested by a consideration of the various particulars in regard to the disease itself, or its special epidemic prevalence:—Short Fever; Fever of Short Periods; Five or Seven Days' Fever; a Five Days' Fever, with Relapses; Remittent Fever; Febris Recurrens; Das recurirende Fieber; Fièvre à Rechute; Synocha; Relapsing Synocha; Inflammatory Fever; Mild Yellow Fever, Remittent Icteric Fever, Bilious Relapsing Fever; Gastro-hepatic Fever; Famine Fever; Die Hungerpest; Fever of the New Constitution; Miliary Fever; Typhinia; Epidemic Fever of Scotland, or of Ireland; and according to the particular years in which it prevailed, as of 1843–44, 1847–48.

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[1] Murchison, loc. cit. p. 295.

[2] A Treatise on Fever, or Selections from a Course of Lectures on Fever, by Robert D. Lyons, M.B. T.C.D. &c. London, 1861. P. 103.

[3] Account of the Epidemic Fever of 1847-48 in Edinburgh. Edinburgh Medical and Surgical Journal, No. 177. 1848.

[4] Notes on the Epidemic Fever of 1847-48. Edinburgh Monthly Journal of Medical Science, vol. ix. 1848.

[5] The disease is well described in Dr. Tweedie's Lectures on Fevers. London, 1862.

Geographical Distribution.—From the foregoing historical statement, it will have been gathered that in Ireland and Great Britain, the former more especially, while in Scotland more than in England, epidemics of Relapsing Fever have chiefly occurred. But not only has this form of fever been more prevalent in Ireland than elsewhere, but it has been clearly shown by Drs. R. Paterson,[1] Wardell,[2] Ormerod,[3] and more recently, and still more decidedly, by Dr. Murchison,[4] that the Irish resident in Great Britain suffers in greater proportion than either English or Scotch from Relapsing Fever. Notwithstanding the statement now made, it is to be borne in mind that the epidemic of 1843 was essentially a Scotch fever, originating in Scotland, and showing little tendency to extend to any great distance. Beyond Great Britain and Ireland, Relapsing Fever has been seen in the Silesian provinces of Prussia and Austria; while an able reviewer, in noticing the account of the Silesian fever of 1847, by Virchow—deputed by the Prussian Government to investigate it—Bärensprung, Dümmler, and Suchanek, has drawn a most interesting parallel between the Irish and the inhabitants of Upper or Prussian Silesia.[5] "During the summer months of 1855," writes Dr. Lyons, "this form of disease was pretty common amongst the British troops in the Crimea."[6] It was not fatal, for the same author observes, "No fatal cases came under our observation in the Crimea." Of its existence in Russia we have recently become aware, for the late exaggerated reports regarding the prevalence of a deadly pestilence in that country and Siberia, have now been shown to point to the occurrence of the Febris Recurrens, or Relapsing Fever of Ireland and Great Britain, associated, as heretofore in our home experience of it, with Typhus, and, probably, also with Enteric Fever. Finally, as regards America, it is evident from the analysis of fifteen cases of Continued Fever, characterised by Relapses, by Dr. Austin Flint, that Relapsing Fever has been observed in the Western hemisphere, but the information supplied by that able writer does not permit us to conclude that this form of fever has ever originated in America; the cases detailed by him may have owed their occurrence, as the much larger numbers occurring in Canada undoubtedly did, to the Irish immigration. Dr. Flint's own statement is as follows: "The conclusion seems unavoidable, that the cases of fever, characterised by relapses, among those which came under my observation in 1850-51, presented the distinctive traits attributed to Relapsing Fever, sufficiently marked to entitle them to be ranked in the class of cases which have been described by different observers as a peculiar form of continued fever."[1]

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[1] Loc. cit.

[2] Loc. cit.

[3] Clinical Observations on the Pathology and Treatment of Continued Fever, by E. L. Ormerod, M.B. London, 1848.

[4] Loc. cit.

[5] The British and Foreign Medico-Chirurgical Review, vol. viii. London, 1851. Article, Diagnosis of Fevers, p. 29.

[6] Lyons, op. cit. p. 106, also p. 108. See also Relapsing Fever, in Science and Practice of Medicine, by William Aitken, M.D. vol. ii. London, 1864.

ETIOLOGY.—Relapsing Fever affects persons of both sexes and all ages; the statistics of the London Fever Hospital, quoted by Dr. Murchison, making it probable "that the proportion of the young to the aged is greater than in the case of typhus."[2] The special season of the year has little, if any, influence on the prevalence of Relapsing Fever, neither is there proof of any particular occupation or employment predisposing to the disease. This much, however, has long been known; and the observation of Relapsing Fever when more recently epidemic in England and Scotland, as well as in Ireland, has strengthened the belief that this disease is peculiarly the fever of the vagrant and the unemployed. The contagious nature of Relapsing Fever scarcely admits of doubt. Two eminent authorities, and these only, have expressed the opinion that this form of fever is non-contagious. One of them, Dr. Craigie, has, indeed, almost admitted the contagious nature of Relapsing Fever: "This," however, he adds, "is rather a presumption than a well-founded inference." The other non-contagionist is Virchow; but, as Dr. Murchison has shown, the importance justly attached to the opinion of this eminent observer is necessarily diminished from the consideration that his experience of the disease was limited to a single fortnight; he left Berlin on the 20th February, 1848, and returned to it on the 10th of March. An examination thus conducted must have been cursory and incomplete. The opinion, moreover, expressed by Virchow, was not shared in by the other medical men of Silesia; all engaged in practice there believed the epidemic malady to be contagious. In all the epidemic visitations of Relapsing Fever, to which reference has already been made, but more especially in those of 1817, 1818, and 1819, of 1843-44, and 1847-48, precisely the same facts which have been held as sufficient to establish the contagious nature of such diseases as Typhus, Scarlatina, and Morbilli, were observed. Physicians engaged in the daily observation of the epidemic fever for many months together, unanimously formed the opinion that the Relapsing Fever propagated itself by contagion. Concerning the earliest mentioned of these epidemics, we find Dr. Welsh writing as follows: "When acting as clerk to Dr. Hamilton in the Royal Infirmary, in the course of four months, my three colleagues, two of the young men in the apothecary's shop, two housemaids, and thirteen or fourteen nurses, caught the disease, and the matron and one of the dressers died of it. Since I left the infirmary, three more of the gentlemen acting as clerks, one of the young men in the shop, and many more of the nurses, have caught the infection, but the number I do not know. In this hospital (Queensberry House), since it was opened on the 23d of February, 1818, my friends, Messrs. Stephenson and Christison, the matron, two apothecaries in succession, the shop-boy, washerwoman, and thirty-eight nurses have been infected ; four of the nurses have died. With the exception of two or three nurses who have been but a short time in the hospital, I am now the only person in this house who has not caught the disease, either here or at the infirmary, within the last eight or ten months. Several students, whom curiosity led too near the persons of the patients, might be adduced as additional evidence. When it begins in a family, we always expect more than one of them to be affected ; I could mention instances of four, five, six, and seven, being sent to the hospital out of one family ; eight, nine, and ten, out of one room ; twenty and thirty out of one stair; and thirty and forty out of one close ; and this all in the course of a few months."[1] The contagious nature of the epidemic fever of 1843-44 is thus insisted on by Dr. Wardell : "Most of the medical officers connected with the Edinburgh Royal Infirmary and additional fever hospitals were seized with it ; eight of the resident and clinical clerks in quick succession became affected, and, out of that number, no less than six were yellow cases, and thus, obviously, in danger of their lives. The majority of the nurses and domestics took the disease, and of the former at one time no less than nineteen were labouring under it. Some of the dispensing physicians and other practitioners took the disorder, as also several of the clergy, and visitors of the sick, whose duties brought them to the bedsides of the patients. The few cases occurring amongst the higher classes, resident in the new town, were generally to be traced to the influence of contagion, the parties affected having had either immediate or indirect communication with those suffering under the disease."[2] And no less decided is the testimony borne by Dr. Cormack : "The disease," he remarks, "is contagious. Of this we have sufficient evidence in the fact that almost all the clerks and others exposed to the contagion have been seized. Dr. Heude and his successor, Mr. Reid, in the new Fever Hospital ; Dr. Bennett, my successor there ; Mr. Cameron and his successor ; Mr. Balfour, in the adjoining fever house ; as well as most of the resident and clinical clerks in the Royal Infirmary, have gone through severe attacks during the past summer and autumn. Hardly any of the nurses, laundry-women, or others, coming in contact either with the patients or their clothes, have escaped ; at one time there were eighteen nurses off duty from the fever ; and of those who have recently been engaged for the first time, or of those who have hitherto escaped, one and another is, from time to time, being laid up."[3] It is in language closely resembling that employed in the sentences now quoted, that Drs. Paterson, W. Robertson, and other physicians have expressed their belief in the contagious property of the Relapsing Fever of 1847-48; and the writer, whose position as resident medical officer in the Fever Hospital while under the care of Dr. Robertson, in the spring months of 1847, afforded him the best opportunity for studying the nature of the epidemic, arrived at the conclusion that the Relapsing Fever, like Typhus, is capable of communication from the sick to the healthy; that, for this purpose, actual contact with the sick is not necessary, the subtle poison of this form of continued fever, equally with that of typhus, being readily conveyed through the air surrounding the latter; and, lastly, that, by means of fomites or clothes, the disease may readily be propagated. It appears sufficiently remarkable that, as specially noted by Dr. Cormack in 1843-44, laundry-women engaged in washing the clothes of the sick, though never brought into direct communication with patients themselves, suffered frequently from the disease: but this, too, was noticed in regard to epidemic cholera; and it was an experience of precisely the same nature in regard to that disease, acquired in the same building, the New Fever Hospital of 1843-44, being in 1853-54 used as a cholera hospital, that, more than anything else, convinced the writer of the contagious nature of epidemic cholera.[1] Resembling typhus in the mode of its propagation by contagion, there is one particular in which these forms of continued fever remarkably contrast. An attack of typhus, for the most part, secures the individual who has thus suffered from subsequent attacks. It is otherwise with Relapsing Fever; no such immunity is by it secured. Welsh noticed, in regard to the epidemic of 1817-18, that "being once affected with the disease seems to afford little, if any, protection against a second, or even a third attack, and that, too, in the space of a few months. I have seen many instances of a second attack within the last twelvemonth."[2] It is well known to the many personal friends, as well as the professional brethren of Dr. Christison, that he has frequently suffered from attacks of continued fever, and we have his own authority for stating that, during the epidemic described by Welsh, he experienced three separate attacks within a period of fifteen months. Dr. Wardell, Sir W. Jenner, and many other writers on Relapsing Fever have noticed the like circumstance. It requires little acquaintance, however, with Relapsing Fever, and but slight familiarity with the remarkable epidemics to which reference has been made, more especially the last two, those of 1843-44 and 1847-48, to feel assured that contagion, while undoubtedly explaining in part the progress of the disease, does not do so wholly, and stops far short of satisfactorily accounting for many of the phenomena which were observed, and this more especially at the very commencement of the different outbreaks.

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[1] Clinical Reports on Continued Fever, based on an Analysis of One Hundred and Sixty-four Cases, &c. &c. by Austin Flint, M.D. Philadelphia, 1855. P. 374.

[2] Loc. cit. p. 303.

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[1] Welsh, loc. cit. p. 45.

[2] Wardell, loc. cit.

[3] Cormack, loc. cit. p. 115.

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[1] Short Account of the Cases treated in the Cholera Hospital, Surgeon's Square, during the late Epidemic, by J. Warburton Begbie, M.D. Edinburgh Medical and Surgical Journal, 1855, p. 253.

[2] Welsh, loc. cit. p. 46.

As assisting the better understanding of a subject still encompassed with difficulty, the generation of fever, there is, in regard to Relapsing Fever, at all events one consideration of very considerable importance. It cannot, we think, be denied that an intimate connexion subsists between this form of fever and destitution. Dr. Alison—who, in all his many writings on fever, as consistently held as he ably supported the doctrine, that intercourse with persons already sick of the disease is the only exciting cause of continued fever, of the efficacy of which we are certain—was evidently greatly impressed by what he and many other observant physicians had noticed of the connexion between destitution, or famine, and fever, in the epidemic of 1846-47 more especially; and he has left it on record as his deliberate opinion regarding the prevalence of fever at that time, that "although burdening the infirmary more than any other which I recollect, it has not for many months spread to any formidable extent among the working classes of the city, but is to be regarded as merely the effect of the unavoidable connexion of this country with the destitution of Ireland."[1] Dr. Murchison has adopted an excellent method of demonstrating the intimacy of the relationship between Relapsing Fever and destitution; he has examined the records of the London Fever Hospital, and shown that since 1847, 430, or 97·5 per cent. of the patients were paid for by the parochial authorities, and totally destitute. Nine of the remaining patients were admitted free, and were also destitute. Not a single patient had been a servant in a private family, and in only one instance was a fee for admission paid by the patient's friends. A large proportion of the patients for some time previous to their attack had been literally starving. Irish writers, with few exceptions, have insisted on the intimate connexion which exists between fever and famine. Stokes described the fever of 1826 as "famine fever," and the well-known pamphlet of Dr. Corrigan, concerning the fever of 1847, had for its title, "On Famine and Fever, as Cause and Effect in Ireland."[2] These physicians, in their respective accounts, referred to epidemics largely composed of Relapsing Fever. Again, the expressions, "Die Hungerpest" and "Famine Fever," clearly indicate that by German observers, as well as by physicians among ourselves, this relationship has been noticed. The whole subject of the etiology of Relapsing Fever has been ably treated by Dr. Murchison, and the opinion which he has expressed of Relapsing Fever being the result of destitution, while typhus is produced by overcrowding and destitution combined, will, we are disposed to believe, stand the test of further observation and renewed careful investigation.

Reference has already been made to the circumstance that at the commencement of the Epidemic Fever of 1843, the disease was speedily recognised as presenting remarkable characters.

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[1] Observations on the Famine of 1846–47.

[2] Dr. Henry Kennedy's Observations on the Connexion between Famine and Fever in Ireland and elsewhere (Dublin, 1847), contain much important information and ingenious argumentation, but, notwithstanding this admission, we adhere to the statements made in the text.

"The present epidemic," wrote Dr. Henderson, to whom is justly due the merit of having first expressed the opinion that Relapsing Fever is a separate and distinct disease from other forms of continued fever, "began to prevail in February last, and the very first cases which fell under my notice I distinguished at once as widely different from every fever that I had formerly seen."[1] About the same time other observers, and more particularly Dr. Cormack, from their separate and independent observation, were led to a similar conclusion; and it is indeed impossible for any attentive reader of the descriptions given by Henderson, Cormack, Wardell, Halliday Douglas, William Robertson, Paterson, Mackenzie, Reid, and more recently, but more particularly, Sir W. Jenner, in which the whole proof is admirably handled and exposed—without arriving at the conclusion that Relapsing Fever is a form of continued fever, wholly different from typhus fever, with which it had formerly been confounded. These observers—and all who have had the opportunity of carefully studying the two fevers, must admit the accuracy of their statements—pointed out that the one fever under no circumstances gave rise by communication to the other, and that an attack of typhus never conferred immunity from Relapsing Fever, any more than the latter afforded protection from typhus. It cannot be considered as offering any serious objection to the view which has now been expressed, that, as in the experience of Dr. Henry Kennedy of Dublin, in 1847-48, cases of Relapsing Fever and typhus have been occasionally met with among the members of the same family, and in individuals occupying at the time of their occurrence the same apartment. Dr. Murchison, indeed, alludes to such, as an occasional experience in the London Fever Hospital, since the well-known observations of Sir W. Jenner were made. The circumstance of the existing epidemic being composed of both forms of fever, and the further circumstance that both fevers are of an infectious nature, satisfactorily explain these coincidences. And, upon reflection, we feel constrained to acknowledge that were the one form of fever capable of producing the other, or, in the view of the spontaneous origin of fever, were typhus capable of being originated in the same way, or under precisely the same circumstances as Relapsing Fever; then the association of the two fevers in the one family, and in the one room among the poor, would have been of infinitely more frequent occurrence than it has ever been proved to be.

SYMPTOMATOLOGY.—The suddenness of its invasion is characteristic of Relapsing Fever. The patient is seized with coldness and rigors, accompanied by headache, pain in the back, and loss of strength. The muscular feebleness and general prostration, however, are not at the first great; for, as Dr. Cormack has observed, "many walk long distances from the country to the hospital, especially during the first days of the disease; and a still greater number of the destitute town patients lounge about the streets after their seizure, and come into us on their legs."[1] The feverishness gradually increases, while the muscular and articular pains and headache become more severe. By the third day, there is usually some amount of epigastric uneasiness, and not unfrequently vomiting. No general abdominal tenderness, however, presents itself, and diarrhœa is of rare occurrence. A perspiration, marked in character, and general over the body, occurs sometimes very early in the disease, on the second or third day, bringing with it little or no relief to the headache and other symptoms. It is from this circumstance, but particularly from the still better marked though more rarely-occurring alternation of rigors and sweating in the earlier days of the illness, that the resemblance to an intermittent fever of irregular character has been remarked by various physicians. On the third or fourth day the fever is at its height, and the case is for the most part characterised as follows:—By greatly augmented temperature of the surface (noted at 102° by Halliday Douglas; as high, and this is very high, as 107° by Wardell); a very quick pulse (this it was which first struck Dr. Henderson as remarkable, unlike what he had witnessed in typhus), very rarely below 100, often 120 (125 on the fifth day, being the average frequency in thirty-eight cases noted by Henderson); it has, however, been noted at 140, and even 160. It was soon determined that, unlike what holds true of typhus, this rapidity of the pulse did not indicate the existence of danger.

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[1] On some of the Characters which distinguish the Fever at present epidemic from Typhus Fever, by W. Henderson, M.D. Edinburgh Medical and Surgical Journal 1844.

With these, the true symptoms of pyrexia, are at the same time associated very slight disturbance about the head, headache frequently, rarely delirium, hepatic and splenic tenderness, with vomiting, great restlessness, thirst, and a white condition of the tongue. In a considerable proportion of the cases a peculiar yellowness of the skin becomes noticeable, best marked in the face, styled by Cormack "facial bronzing," and to this a distinct jaundice, with urgent vomiting, sometimes succeeds. To these symptoms there occurs, usually on the fifth or seventh day, an abrupt cessation. Nothing can be more remarkable than the sudden change—usually ushered in by a profuse perspiration, less frequently by an epistaxis, or other hæmorrhage, or by diarrhœa—effected in the condition of the patient. The frequent pulse and hot skin have in a few hours vanished, there is a normal appearance presented by the tongue, and, as Cormack has described it, "one day we hear the patient moaning and groaning in pain, and on the next he is at ease and cheerful, his only complaint being of hunger and weakness." The condition of apyrexia established, the patient continues to improve; he gains strength, often rapidly, and convalescence appears to be altogether satisfactory, except that the pulse sometimes continues remarkably slow. On or about the fourteenth day from the commencement of the original attack the relapse takes place; there occurs a second paroxysm of fever in all points similar to the first; it may, however, be more severe, or on the contrary less severe in its symptoms. The duration of the relapse is usually three days; it may extend to five days, or even longer, and, when usually mild, it may terminate before the third. A second relapse, usually occurring about the twenty-first day, is far from uncommon. "Not less than five of these accessions or 'relapses' have been known to occur."[1]

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[1] Loc. cit. p. 3.

To some of the more remarkable phenomena now briefly alluded to, and to a few other features in the symptomatology, it is proper to direct attention a little further in detail. Relapsing Fever, properly speaking, is undistinguished by cutaneous eruption. The most careful observers have failed to notice in it the measly rash so characteristic of typhus, and no one has described the rose-coloured spots (*taches roses lenticulaires* of Louis) now regarded as equally characteristic of enteric or typhoid fever. The "measly-looking efflorescence" noted by Welsh in 1819, as occasionally present, may fairly be considered to have occurred alone in the cases of true typhus which constituted a portion of the epidemic he observed. Petechiæ, hæmorrhagic spots, and vibices have all been described as of occasional occurrence, while Dr. Ormerod found a miliary eruption (sudamina) so common in the Relapsing Fever of 1847, that in his description he gave to the disease the name of "Miliary Fever."[2] Dr. Halliday Douglas found sudamina very rarely in the fever of 1843–44, and the writer can answer for their presence in that of 1847 being likewise quite exceptional. But another cutaneous appearance, although variable, is of decided importance, —although it is probable that its gravity as a symptom has been unduly estimated by some physicians,—namely, a yellowness, or jaundiced hue. Welsh also noticed this: "a yellowish, dusky state of the skin was not unfrequently observed." In connexion with it he likewise noticed that "the patient's urine distinctly tinged linen cloth or similar substances immersed in it."[3] In the epidemics of 1843 and 1847 jaundice was observed, in the former specially by Cormack, Wardell, and Douglas; in the latter by Jenner, in London; also, but with greater rarity than during the previous epidemic, in Edinburgh, by William Robertson and R. Paterson.

Nausea and vomiting have been described as common symptoms in Relapsing Fever. The matters vomited have usually been found to consist of the ingesta, frequently tinged with bile. Occasionally an appearance resembling the black vomit of yellow fever was noted, as by Cormack and Wardell, who regarded it as a peculiarly unfavourable sign, and by Dr. Arnott of Dundee, who, looking upon black vomit as quite common in its occurrence, did not find it by any means a fatal indication. Peculiarities, as regards the appearance presented by the tongue, have been noted. It is usually from the commencement coated with a white or yellowish fur, while a small triangular space towards the point of the tongue, as well as its edges, is clean, and often redder than natural. In mild cases the tongue continues moist throughout the attack; but in the more severe, dryness, blackness, and incrustation with sordes, occur. The appetite suffers in Relapsing Fever for the most part, as in other febrile disorders, but many observers have stated that an unusual and sometimes altogether inordinate desire for food has distinguished particular cases. The urine in Relapsing Fever was specially examined by Dr. Henderson and Mr. (shortly afterwards Dr.) Michael Taylor. Occasionally the quantity is reduced, or there may even be suppression of the secretion, while the amount of urea is greatly diminished; in connexion with these changes the occurrence of serious cerebral symptoms is to be apprehended. Here our knowledge of a very important topic ceases. Dr. Parkes has truly observed that scarcely anything definite is known on the subject.[1] Dr. Henderson had his attention early directed to the condition of the kidneys, and satisfied himself that the occurrence of convulsions, coma, and less serious symptoms of a nervous nature, were to be ascribed to interference with the proper function of these organs. In connexion with a diminished amount of urea in the urine, Dr. Douglas Maclagan determined, by investigations undertaken at Dr. Henderson's request, the existence of an increased amount of urea in the blood.

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[1] British and Foreign Medico-Chirurgical Review, vol. viii. p. 8.

[2] Clinical Observations on the Pathology and Treatment of Continued Fever, from Cases occurring in the medical practice of St. Bartholomew's Hospital, by Edward Latham Ormerod, M.B. London, 1848. P. 217.

[3] Loc. cit. p. 21.

There are certain complications and sequelæ of Relapsing Fever. The inflammatory affections within the chest which are known to occur in the course of the other forms of continued fever—at times seriously influencing the mortality which these occasion—are met with also in Relapsing Fever; bronchitis, broncho-pneumonia, pneumonia and pleurisy. Laryngitis, requiring tracheotomy, occurred in one case in the experience of Dr. Paterson. The writer remembers that the presence of a similar inflammation necessitating the same operation in at least one other case, was ascribed, at the time of its occurrence in 1848, to the patient having been peculiarly exposed to cold while under treatment in one of the temporary sheds erected for the accommodation of the fever patients admitted to the Royal Infirmary of Edinburgh. Hæmorrhages of various kinds have been noted to occur, for the most part, about the period of crisis. Paralysis of a local nature—of the deltoid muscles, as observed by Cormack—and much more frequently severe muscular and articular pains, are among the number of the nervous complications which have been observed. Parotitis, which is familiar to us in typhus and enteric fever, and other glandular enlargements and suppurations, were certainly of uncommon occurrence in the epidemics of Relapsing Fever witnessed in this country. In the recent Russian epidemic it would appear that these buboes are more frequent; and Mr. Simon has no doubt correctly inferred that "from this circumstance there arose the rumour of plague."[1] Diarrhœa, sometimes taking the place of perspiration, has been described by many observers as the critical evacuation in Relapsing Fever; with a greater degree of frequency it occurred during the relapse, or after recovery from the relapse. That diarrhœa raised the mortality considerably, is evidenced by the statements of various of the Scotch physicians.

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[1] The Composition of the Urine in Health and Disease, and under the Action of Remedies, by Edmund A. Parkes, M.D. London, 1860. P. 260.

There are few more interesting circumstances known in regard to Relapsing Fever than the frequency with which pregnant women abort or miscarry. By some this accident has indeed been described as invariable; it is not so, but the exceptions are infrequent. In relation to this peculiarity, Dr. Murchison has truly observed that, on the supposition that Relapsing Fever is but a mild variety of typhus, it would be very remarkable that, in the former, abortion is almost invariable, and the fœtus dies; whereas, in the latter, abortion is the exception, and when it occurs, the child, if near the full time, usually lives.[2] A sudden, wholly unlooked-for, and at times fatal, syncope has distinguished some cases of Relapsing Fever. Dr. Halliday Douglas mentions one such in which the patient was found dead about the period of the first crisis, and only half an hour after she had expressed herself as feeling easy. An occurrence of this kind, though fortunately not common, may well be considered important in a prognostic point of view. Lastly, an interesting form of ophthalmia presenting two distinct stages, the amaurotic and the inflammatory, has been met with and specially described by Dr. Mackenzie of Glasgow, as post-febrile ophthalmia.[3] In that city so frequent was the eye affection in 1843, that Dr. Andrew Anderson speaks of multitudes of cases having been treated at one Eye Infirmary.[4]

When the frequency of the occurrence, either of more than one relapse, or one or other of the complications or sequelæ which have been attended to—others less frequent and of less severity it has been thought unnecessary to mention—is considered, it will be understood that Relapsing Fever, though happily occasioning a mortality greatly inferior to typhus, is a fever determining—in not a few of the sufferers from it—long-continued bodily weakness, while in a still larger number convalescence is greatly protracted.

The account which has been given of the symptoms and course of Relapsing Fever is sufficient, we think, to establish its separate and distinct nature, and to allow its being readily distinguished from typhus, enteric fever, febricula, remittent, or yellow fever, diseases with the majority of which it has been at one time or other confounded. The relapse, which is the distinguishing feature of this pyrexia, is, properly speaking, unknown in any other form of fever; and Sir W. Jenner's careful observations, confirmed by the experience of many competent authorities, have proved, that Relapsing Fever—a contagious disease as we have already seen—is capable only of giving rise to a similar disease; it can engender no other form of fever, and no other form of fever can engender it. What there is in the morbid anatomy of Relapsing Fever, and in the mortality it occasions, to sustain and confirm this opinion, we shall now inquire. As to the former, unlike what holds true of pythogenic or enteric fever, there is then no constant or invariable morbid appearance to be detected. Nevertheless, there are a few thoroughly ascertained facts in regard to this subject, which, in the not unlikely event of another occurrence of Relapsing Fever, should form the groundwork for renewed and still more extended investigation. The spleen is almost always found altered; enlargement and softening, nearly in some instances to the extent of diffluence, are the most frequent changes, but increased firmness in its structure, and fibrinous deposits in the splenic substance, have likewise been observed. Enlargement and engorgement of the liver, without any structural change—even as has specially been stated by Cormack and others—in the best marked "yellow cases," have been generally found. The blood, when subjected to microscopic examination, has revealed the existence of an increased number of white corpuscles, similar to what occurs in the now better understood condition of leukæmia.

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[1] See his letter to the Lord President of the Council, dated Whitehall, April 19, 1865. It may be right to mention here, that in the experience of the writer, parotitis, hitherto a very rarely observed phenomenon by him in fever, has during the last twelve months been seen frequently, both in typhus and enteric fever.

[2] See a confirmation of the last observation in an account published by Dr. Matthews Duncan, of a case which recently fell under the writer's notice. Edinburgh Medical Journal, September 1863.

[3] See Mackenzie, in Medical Gazette, 1843.

[4] Lectures on Fever, p. 135.

The mortality occasioned by Relapsing Fever is usually not great, being far inferior to what is commonly observed in either typhus or enteric fever. In the recent Russian epidemic, Relapsing Fever is, according to Mr. Simon, "causing more than its usual proportion of deaths;" but that usual proportion does not exceed 4·75 per cent., or one death in every twenty-one persons attacked. Dr. Murchison, on placing together the results noted at the London Fever Hospital, with those detailed by various physicians during the Scotch and Irish epidemics, has given as the total 14,119 cases with 672 deaths—yielding the rate which has just been quoted. Age and habits influence the mortality in a manner closely resembling that which is witnessed in typhus and adynamic fevers generally. The mode of fatal termination is not always alike; the occurrence of a sudden and fatal syncope has already been noticed; and, as originally indicated by Dr. Henderson, the impaired action of the kidneys, leading to the imperfect elimination of urea, causes a death by coma. An asthenic termination, too, may occur in those instances of the disease in which one or other of the complications already adverted to, have manifested themselves.

THERAPEUTICS.—If, as we believe, there is an intimate connexion between famine—by which is understood poverty and destitution—and

Relapsing Fever, then it will readily be conceded that such attention to the wants of the poor, particularly as regards due nourishment, as ameliorates their condition, will tend directly to prevent the origin and to arrest the spread of this disease. It is likewise contagious, and therefore isolation of the sick should, as much as possible, be secured. Dr. Welsh, to whose account of the epidemic fever of 1817 and 1818 we have frequently had occasion to refer, conceived that blood-letting was the great remedy. Little reliance, however, can be placed upon the therapeutic observations of Welsh, seeing that neither he nor other physicians of his time had distinguished between typhus and the fever which showed the tendency to relapse. Further, it will, we think, appear to any attentive reader of Welsh's interesting work, that the changes in the condition of the patient, particularly the reduction of the pulse, the diminution of the fever heat, the occurrence of sweating—ascribed to the beneficial operation of the blood-letting —were in reality merely those changes which a subsequent better knowledge of the disease has led us to recognise as the essential phenomena of the fever itself, invariably occurring in its course, and uninfluenced by any treatment. All attempts to ward off the relapse, and for the most part attempts to postpone it, have signally failed. Those antiperiodic remedies in the use of which we place reliance, and the virtues of which in the ordinary intermittent and remittent fevers have been incontestibly established—such remedies as quinine, arsenic, bebeerine, salicine—have all been faithfully tried, and all have failed.

Emetics of ipecacuanha, or of ipecacuanha with antimony, and mild laxatives, exhibited early in the disease, have appeared to be useful in the hands of various physicians in relieving portal congestion, and producing freedom from the oftentimes distressing pain, or at least uneasiness experienced in the region of liver, stomach, and spleen. Diuretic remedies—and particularly the salts of potash—were serviceable in relieving the tendency to head symptoms, by which as we have already stated, some cases of Relapsing Fever were distinguished.

Those considerations for the proper employment of food, and the administration of stimulants, which should guide the physician in the treatment of the other forms of continued fever, are available likewise in the instances of Relapsing Fever. It seems unnecessary to enter on an analysis of these here; and while the complications and sequelæ of Relapsing Fever are, as we have seen, sufficiently remarkable, there is only one, the ophthalmic affection, the treatment of which seems to require a brief description; this may be given in the words of Dr. Anderson: "We learned very important lessons from the treatment of this ophthalmia—lessons which tell against some of the theories which are fashionable at the present day. The previous fever and the actual debility of the patients made us at first eschew anything like depletion; but we found on the failure of other means that bleeding was the most effectual—the only effectual—mode of cutting short this dangerous ophthalmia."[1] After the abstraction of blood, calomel and opium were administered, until there appeared evidence of the system being slightly affected by the mercury. These remedies may be prudently combined with quinine and a generous diet, while it is almost unnecessary to add that in the treatment of the purely and simply amaurotic affection, altogether unconnected with hyperæmia, which occurs as a sequela of Relapsing Fever, only tonic remedies and an invigorating diet are required.

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[1] Anderson, op. cit. p. 135.

YELLOW FEVER.

By John Denis Macdonald, M.D. F.R.S.

Definition.—Infectious continued fever, ushered in with languor, chilliness, and more or less severe lumbar pain and frontal headache; countenance flushed; eyes at first humid, then suffused, and ultimately ferrety; skin imparting a tingling heat to the touch, and as the second stage advances, gradually acquiring a lemon or greenish-yellow tinge; mind usually disturbed with hallucinations, or more or less violent delirium; restless watchfulness, or, possibly, drowsiness, even to extreme coma; epigastric uneasiness; spontaneous vomiting, without effort, first of a clear glairy fluid, but subsequently with "coffee-ground" flocculi, or blood itself, often, towards the close, with irrepressible hiccough, and wild shrieking or melancholy wailing; tendency fatal, but the disease generally confers an immunity from subsequent attacks.

Synonyms.—Yellow Fever, Bulam Fever, Hæmagastric Pestilence, Black Vomit. *Latin*—Febris Flava, Synochus vel Typhus Icterodes, Synochus Atrabiliosa. *Spanish*—Fiebre ò Calentura Amarilla Vomito-negro ò Prieto. *French*—Fièvre ou Typhus jaune, Fièvre Mattilote, Mal de Siam. *Italian*—Febbre Gialla.

History.—From the year 1647, when the first recorded outbreak of Yellow Fever in the West Indies[1] occurred, to the present time, this disease has been recurring at irregular intervals in the epidemic form, and gradually extending its range. It has, moreover, appeared, in many instances, to borrow new vigour by its importation from one place to another, and though it may be said to be permanently present in some localities, *e.g.* the islands of St. Thomas and St. Domingo, there is no proof whatever of its spontaneous development anywhere. Every epidemic seems to have an assignable source, and even where this is not very obvious, there are *à priori* reasons enough to trust that it would be discoverable, were investigation properly instituted.

The symptoms of Yellow Fever manifestly result from the more or less potent operation of some subtle organic poison upon the system through the medium of the blood; and the very fatal tendency of the malady is probably linked with nature's efforts to eliminate the poison by the gastro-intestinal mucous membrane, when both liver and kidney have been rendered ineffective in bringing about this result. The fact may want confirmation, but it has always occurred to the writer, that the liver is especially active in persons perpetually exposed to the specific infection of the disease, without, however, yielding to its influence; as though the elimination of the poison had been effected, in their case, without developing the train of symptoms proper to the disease.

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[1] Ligon's History of Barbadoes.

Whatever physical conditions—such as increase of temperature, moisture, and subsequent evaporation, and the like—may be favourable to, or merely coincident with, the development of Yellow Fever on shore, when once communicated to a ship, and isolated by far removal from all local influences, its phenomena are very striking and suggestive. Under such circumstances it is difficult to witness the spread of the disease from one individual to another, and its virulence becoming more intensified by the unavoidable crowding of the sick, without recognising the important part which the emanations and excretions of the human body itself must take in the matter. It may be objected that all the most potent of the terrestrial or atmospheric conditions alluded to are fulfilled in the bilge effluvia; but as these, *per se*, have never been known to originate the disease, we are driven to one or other of two positions, either that they have nothing to do with it, being simply coincident like other things that might be named, or that they form the nidus for its further development and spread, subordinate to a specific cause. It must, however, be apparent to a close observer, that the human element far outweighs all other suppositions, and, in this point of view, the refinement and subtlety of the poison may be more easily conceived. If Yellow Fever be referrible to the zymotic class, in support of which idea several cogent arguments might be adduced, its mode of origin cannot be materially different; and few nowadays would attempt to trace the whole of the specific virus of rubeola, scarlatina, or other true exanthemata, to any of the common decompositions of cesspools or the effluvia evolved in bad drainage, and such, otherwise, certainly unwholesome conditions.

We are in want of proof of the spontaneous development of Yellow Fever independently of infected places or persons, while a knowledge of the precise nature of its specific cause is still a desideratum to medical science. But that it is endemic in certain localities, though varying much as to its intensity, and the periods of its manifestation, cannot be doubted; for under similar circumstances, *i.e.* of latitude, or climate, &c., certain other localities have never been visited by the disease.

What has just been said in regard to Yellow Fever very strikingly points to analogous facts in the geographical distribution of plants and animals, and the curious laws that regulate both their propagation and decline. But it may be considered probable, however much the human system may modify their composition and manifestation, that the primary zymotic poisons owe their origin to the development of the humbler and more minute, and therefore more subtle, forms of animal and vegetable life; such being always coincident with a corresponding amount of decomposition, and the evolution of new or simply liberated compounds in a gaseous or diffusible form.

The presence of offal and filth, or stagnant water, with infused animal and vegetable matter, may be regarded as affording something more than predisposing conditions; and such a state of things is often, though certainly not always, discoverable where Yellow Fever is rife, but more particularly in sultry weather, after heavy rains.

The periodical occurrence of Yellow Fever, with intervals of immunity, has its parallel in a fact well known to the students of the diatomaceæ and dermidiaceæ—namely, that particular species, which are known to exist in a definite pond or pool one season, may be at another replaced by forms never before detected in the same spot; while, again, the original species, under favourable and often unaccountable circumstances, reappear after the lapse of a certain time.[1]

It is not our intention to make even brief reference to all the views that have been put forward as to the nature of the specific cause of Yellow Fever; but it may be remarked that even if they were carefully detailed, our conclusions would probably be the same; for upon this head a very little more than what has been above stated is critically known.

The infectious nature of Yellow Fever is now not only generally admitted, but it forms one of the most distinctive features of the disease, at once marking it off from those fevers which in nearly every other particular simulate it.

It is scarcely necessary to multiply or repeat the "strong proofs" in this place, after the satisfactory evidence lately brought before the Epidemiological Society of London, in the papers of Dr. Bryson, and the important verbal support of the late lamented Dr. M'William. The tenets of the writer, derived from actual facts, are the following, acknowledging a genealogy to the widely-spread family of Yellow Fever.

1. That the first place or the first person, or both, must have become infected, somewhere or somehow.
2. That by veritable, but unknown, or rather untraced, links with this source, places, having become infected, may infect persons.
3. That persons infected may infect other persons and places previously presumed to be healthy.
4. That the clothing of infected persons, or of healthy persons having communicated with infected places or persons, may impart infection to other places or persons.
5. That if places were moveable, like persons (which is literally true of ships), on being infected, they would impart the virus to other places in sufficiently close proximity.
6. Finally, from the investigation of the history of particular cases, it has been satisfactorily shown that the period of incubation, or

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[1] Med. Journal of H.M.S. *Icarus* for 1860, N. A. and W. I. Station.

latency, in this disease, *i.e.* from the imbibition of the poison to the first appearance of symptoms, ranges from one to fourteen or fifteen days.

ALTITUDINAL AND HORIZONTAL RANGES.—It may be very well to assign an altitudinal limit to the spread of Yellow Fever—and, roughly speaking, this may be estimated at between 2,000 and 3,000 feet above the level of the sea—but the local conditions of every country seem to determine a range peculiar to itself. Thus, the disease has been known at Newcastle, Jamaica, at an elevation of 4,000 feet; while in the Valley of the Mississippi its highest recorded range is about 600 feet (admitting the Fever of Gallipolis to be of the genuine type). Humboldt alludes to the Farm of Encero, in Mexico, at an elevation of 3,243 feet, as the altitudinal limit of Black Vomit. At St. Domingo, the mountain encampments of the French in 1792, and of the English in 1796, enjoyed an immunity from the disease, while it was spread far and wide amongst the troops in the low country.

Though the West Indian Islands, and the neighbouring coasts of North and South America, may be looked upon as the focal area of Yellow Fever, yet, taking the outlying points at which its occurrence in the epidemic form has been recorded, its geographical range must be regarded as very considerable indeed, *i.e.* between 97° west and 2° east longitude, and between 48° north and 35° south latitude.

At least for the space of a century and a half, up to the year 1850, the river Amazon, dividing the Brazils from Guiana, limited the extension of Yellow Fever south of the line; and while the disease was raging at Rio and Bahia, at the close of that epoch, the Montevideans flattered themselves that they were without the geographical limit of the pestilence, until it fell to their turn to sustain its visitation several years later, when the illusion was dispelled. Similar facts may be adduced with regard to the extension of the disease along the shores of the Pacific; so that, however well we may be acquainted with its present range, making all due allowance for temperature, we cannot tell what the future may bring forth. In this connexion it may be mentioned that a temperature of at least 72° is assumed to be essential to the development of Yellow Fever, though cases exceptional to this rule also may now and then happen.

SYMPTOMS.—With or without such premonitory symptoms as loss of appetite, costiveness, flatulence, a sense of debility, and the eyes humid and bright, the disease frequently makes its invasion with chills; but this will greatly depend upon the existing temperature or climate. Thus Jackson maintains that they seldom occur within the tropics, while they are quite usual in more temperate climates. The chills alternate with flushes of heat, and the latter gradually settle down into regular fever, which is often observed to become more severe towards evening, with something approaching a remission in the morning. The amount of fever, moreover, bears relation to the severity of the chills preceding it. Frontal headache is also an early symptom, with shooting pains through the orbits and temples; but distressing as these may be, they are usually trivial in comparison with the agony of the lumbar pains which frequently seize the patient at this period, and fell him to the ground in a writhing and convulsive state. In some severe cases, however, this symptom is nearly entirely absent.

From the very commencement the patient may be troubled with nausea and epigastric tenderness, or they may be developed as the reactive stage advances.

The pulse exhibits great diversity of character, being much accelerated, full, and strong, in keeping with the force of the paroxysm, or even soft and weak where the febrile reaction is deficient in severe cases—the beats ranging between 90 and 120 in a minute.

Also varying with the nature of the paroxysm, the skin may be hot and pungent, dry or perspiring, livid, flabby, and even cold.

The tongue exhibits a creamy-white coat on the dorsum, with red tip and edges, and injected papillæ, with or without soreness of the throat.

As the second stage advances irritability of stomach is added to the nausea, and the epigastric pain and tenderness become more distressing. The patient craves for cold drinks, which are immediately rejected, first with some retching and pain, but subsequently without effort. The matters vomited usually have a suspicious appearance: thus, they are sometimes imbued with bile, lightly streaked with blood, or quite serous with small chocolate-coloured flocculi, discovering the tendency to hæmorrhagic oozing from the lining of the stomach.

The urine is scanty, high-coloured, and probably albuminous; the stools become gradually more and more deficient of bile; and the bowels are often obstinately constipated.

The patient begins to be restless and vigilant, and disposed to leave his bed, go into another, or walk about naked if he be permitted. He exhibits an evident derangement of intellect, though he may answer questions coherently. In other instances,—with a suffused ferrety eye and a drunken expression of countenance,—wild hallucinations, similar in every respect to those of delirium tremens, afflict the victim's mind, and may deceive the practitioner most seriously in sporadic cases.

Febrile reaction may continue for an indefinite period between a few hours and two or three days, and its duration is said to be in the inverse ratio of the violence of the attack.

"Having run this course," says Dr. La Roche, "the fever subsides, never more, or very seldom, to return—the disease being one of a single paroxysm—and is followed by a state of remission or metaphosis." The nature of this remission is all important, as regards the fate of the patient. Should all the symptoms be alleviated, the pulse becoming less frequent, or even normal, the delirium subsiding, and, above all, if there is no more irritability of stomach; active diaphoresis, epistaxis, or a critical discharge of bile from the bowels, may place him on the highroad to recovery. Should the skin have assumed its lemon-yellow tint, it will remain all through the convalescence, towards the close of which the writer has noticed a desquamation of the cuticle, much resembling that of the ordinary exanthemata.

If, on the other hand, the ferrety eye whitens, the cheek grows pale, and the lips are blanched, while the pulse is weak and compressible, and the delirium is persistent with irritable stomach, the apparent remission is delusive, and a fatal issue is pending. The patient refuses all medicine and food, lies down very much against his own inclination, cramps gather in the calves of his legs, and while they are being rubbed by the attendants, his delirium becomes frantic, and is probably retrospective of former impressions. His utterance is supernaturally rapid, keeping pace with a panoramic sequence of ideas in which the mind is absorbed, the pulse is imperceptible at the wrist, and just when physical exhaustion has merged into death a final automatic discharge of black vomit closes the tragedy. It is only at this period, in many cases, the lemon-chrome tint of the skin makes its appearance. When it happens in the course of the disease, *i.e.* third stage, it is observed to spread from the forehead downwards to the face, neck, and chest, and then it becomes general.

In another class of cases, the pulse gradually moderates, but thirst increases, and epigastric heat and pain are persistent, with irrepressible vomiting and hiccough, but the mind is calm and coherent to the last, though quite conscious of its tendency to wander. Indeed, the effort of the intellect to correct vagrant ideas, and give them a rational form, is often affectingly observable, more particularly in the case of educated persons.

Contrasting remarkably with the class of cases just described, some persons exhibit a tendency to drowsiness at a very early period of the disease, and finally settle down into a placid state of coma, not unlike that of severe concussion of the brain, without pressure or organic lesion.

This state of things, no doubt, results from uræmic poisoning in connexion with the suppression of the urinary secretion.

It is a fact, worthy of special note, that the heart's action may continue long after the pulse at the wrist has become imperceptible, and when all respiratory movements have ceased.

Death may happen in the course or at the close of any of the three stages of the disease, namely: 1st, the accessionary; 2d, the reactive; or 3d, the remissional: and this will of course be in accordance with the type or variety assumed by the malady.

Further remarks on the symptomatology, rendering the idea here given of it more complete, will be found in the section on the classification and varieties of the disease; as it is of importance to avoid unnecessary repetition.

Diagnosis.—Though a very good general sketch of Yellow Fever may be given by any one who has witnessed an epidemic of the malady, it is not quite so easy to isolate the symptoms that may be fairly assumed to be pathognomonic. Indeed, the whole aspect of the disease is often so diversified, or distinguished by the absence of this or that symptom where the collateral evidence of its identity is indubitable, that it is difficult to say which feature is of most diagnostic importance. Add to this the actual occurrence of the several symptoms of Yellow Fever in some part of the course of other febrile disorders, which, after due consideration, have been declared to be essentially distinct, and the difficulty will be still more apparent.

Inasmuch as yellowness of the skin and conjunctivæ, not merely from jaundice, but from the effusion of the hæmatine of the blood itself, as in Yellow Fever, sometimes occurs in the paludal remittent fevers of various countries, that character, singly, cannot be pathognomonic of Yellow Fever. Of black vomit also the same thing may be affirmed, so that two of the most important features of the malady are scarcely available for a satisfactory diagnosis. Much stress has been laid upon the very constant symptom of frontal headache, in connexion with the watery and suffused eye, the white, creamy, or cottony coat of the tongue, and its red tip and edges; and in particular the early appearance of albumen in the urine; but none of these characters, nor even all together, can be more definite than the following positions:—

1st. Now that there can be no doubt of the infectious nature of the disease, it may thus at once be distinguished from those disorders with which it is likely to be confounded, for this property is sure to be developed in every epidemic of specific Yellow Fever.

2d. As Yellow Fever is one of a "single paroxysm," of longer or shorter duration, and divisible into three stages, the disease is continuous in its type, or it runs a definite course without such remission and exacerbation as are seen in the paludal fever.

3d. If Yellow Fever shall have passed through all its stages without destroying life, it in general confers immunity from a second attack.[1]

The first appearance of Yellow Fever not infrequently presents no other symptoms than those of an ordinary ephemeral fever; for which the writer very naturally mistook the first case of Yellow Fever that ever fell under his observation. The disease, however, declared itself on the second day, and there was no further doubt as to its real nature. There is a still greater possibility of confounding malarial remittents with specific Yellow Fever, and even good observers have regarded them as identical. In this connexion, however, we may appropriately quote Dr. Maclean's bold diagnostics, as given by Dr. Aitken.[2] "I am now myself a firm convert to the doctrine that Yellow Fever is specifically distinct from remittent. To this opinion I have come with a full knowledge of the fact, that some cases of remittent fever in India closely resemble some of the forms of Yellow Fever. But of this I am now certain, that the Yellow Fever of the true yellow fever zone is unknown in India, where true malarial fevers abound. There is in true Yellow Fever, for the most part, an absence of that periodicity which is an unfailing characteristic of true malarial fevers. Then there is the difference, so well insisted upon by Blair, in true malarial fevers. Men do not pass from recovery to health, as is the case in such a marked degree in Yellow Fever, after which there is no, or very little, evidence of the existence of any cachexy. Malarial fevers exist and are destructive at a temperature at which Yellow Fever is at once destroyed. Albuminous urine is almost invariable in Yellow Fever—only occasional in remittent. There is in Yellow Fever an unexampled range of hæmorrhages; in remittent fever these hæmorrhages are often, indeed generally, absent. Quinine has a power over malarial fevers that is beyond the reach of doubt or cavil; the same is not true of Yellow Fever. Men suffer from malarial fevers again and again; second attacks of Yellow Fever are, to say the least, rare."

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[1] In H.M.S. *Icarus*, in 1860, only five persons out of about 130 escaped an attack of Yellow Fever; yet not a single case of relapse occurred during the whole course of the epidemic.

[2] Science and Practice of Medicine, vol. i. pp. 479–80.

PATHOLOGY.—*Temperature of the Body.*—The writer has observed that when patients previously treated in the open air on board ship were transferred to hospital, the body exhibited a marked increase of temperature, and the febrile symptoms became more active. Moreover, any part of the body exposed more than another soon evidenced a diminution of animal heat. In the axilla the temperature may range from blood heat to 107° Fahr., the maximum observed by Dr. Blair. In the "sthenic form," in comparison with others, Dr. Lyons noticed a general elevation of temperature ranging between 3° and 7° Fahr.

Coloration of the Skin.—On the first accession of the disease the skin becomes pale and, perhaps, shrivelled to a greater or less extent; but, as the reactive stage sets in, it warms up and grows red; the face in particular appearing animated and flushed. The depth of this redness, of course, bears relation to the intensity of the febrile symptoms, and it is looked upon as pathognomonic from its very constant occurrence, whenever reaction is developed at all; but should the latter be defective, the countenance may be pale, livid, or sallow.

The so-called jaundicing of the conjunctivæ and skin is by no means a constant symptom, but may be more or less characteristic of particular epidemics or of different stages of the same epidemic.

It is highly probable that the greenish-yellow hue is often due to the presence of bile. But, as I believe was first suggested by Warren,[1] and subsequently by Sir G. Blane and others, the lemon yellow and orange tints are unquestionably owing to the solution and effusion of the colouring matter of the blood. M. Guyon regarded it as nothing more than the tinge of contusion. They should not be confounded with the much darker and more greenish hue of Yellow Remittent Fever, depending altogether upon jaundice, and therefore of a very different nature.

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[1] "This yellowishness, I am persuaded, chiefly arises from a more complete colliquation or dissolution of the red globules of the blood into a yellowish serum, which will naturally soon give that tincture to the whole skin. The same is also observable on human bodies soon after bites of some poisonous serpents, or other venomous animals; and, in such

Though yellowness of the skin may set in at any time from the first to the fifteenth day, or even exhibit itself after death, yet, from Dr. Blair's observations, it occurs most frequently on the fourth and fifth days of the disease. It is important to note, however, that it makes its appearance coincidently with the black vomit in the generality of cases.

The Tongue.—It is only when the febrile reaction is taking place, or even some time after this has begun, that the tongue in general assumes its characteristic white coating, with red tip and edges. Previously to this it may be quite normal in appearance, only perhaps bearing the impressions of the teeth. Indeed, instances occur in which it remains without marked change, even up to the close of the malady, either in recovery or death. The characters of the tongue, therefore, cannot be always said to go hand in hand with the increased heat of skin and acceleration of pulse.

After having first become coated with a white creamy substance, this condenses into a thick cottony fur, and the marginal papillæ become enlarged. The coating becomes thicker towards the base of the tongue, and more discoloured; moreover, one, two, or more yellowish, brown, or black longitudinal bands run down its middle. When hæmorrhage arises from the parts about the mouth, the epithelium of the tongue and fauces is soon stripped off, leaving the surface glazed with half-dried blood and sordes.

Under such circumstances, the tongue is more pointed and smaller than usual, of a mahogany-red colour, and more or less fissured. As might be expected, the denuded throat is sore and requires special treatment.

The accompanying table, as quoted by Dr. La Roche, from the records of Roper Hospital, Charleston, during the sickly season of

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cases, it cannot with any reason be supposed to proceed from a suffusion of bile, but rather from a colliquation, and perhaps a gangrenous diathesis of the sanguineous mass, occasioned by the force of the deleterious venom that had been infused into it. What is observed every day in all common bruises of the flesh may serve somewhat further to elucidate the matter; for here, when the texture of the extravasated blood begins to loosen and dissolve into a liquid serous consistence, a very visible yellowness appears in and about the part; but this soon goes off again, when the matter is fully absorbed back into the vessels, where it commits no hurt, but is readily overcome by the force of nature, as the quantity of such dissolved blood is small, and at the same time very innocuous. I do not, however, deny but that, through a great propensity and straining to vomit, some quantity of the bile may be thrown into the blood; but then I must observe, that the yellowness of this distemper I am speaking of very frequently shows itself when there has been no vomiting or retching at all, or scarce any sensible sickness of the stomach; for the truth of which I can appeal to many."—Treatise on the Malignant Fever of Barbadoes, p. 11, as quoted by Dr. La Roche.

1854, gives a good idea of the difference of character presented by the tongue in the three stages of Yellow Fever.

Condition of the Tongue.	1st Stage.	2d Stage.	3d Stage.	Total.
Swollen	4	29	31	64
Dry	52	23	14	89
Bloody	—	3	31	34
Whitish	44	33	18	95
Brownish	94	53	39	186
Moist	109	109	110	328
Red	43	26	33	102
Velvety and white	23	2	3	28
Black	1	1	19	21
Natural	26	10	7	43
Glazed	4	2	—	6
Cracked	—	3	—	3
Total number of observations				999

The Pulse.—If the tongue has been observed to be variable and inconstant in its character, the same may be said of the pulse, and trusting to it alone a very incorrect prognosis may be formed. Thus, it has been known to preserve an apparently normal state, even coincidently with the most portentous symptoms; and it has been previously noticed that the pulsation of the heart itself may continue some time after all respiratory movements have ceased. It is easy to imagine the evil results that might follow active depletory measures in the reactive stage, when the force of the pulse usually gives so false an idea of the stamina of the system (for, naturally, without such means, the pulse diminishes in force and frequency as the third stage sets in); but their employment in accordance with *primâ facie* indications may render the ebb fatally low.

The pulse in Yellow Fever is usually full; but it is assumed to be less tense and hard than it may be in other fevers at a corresponding stage, and from a frequency of 100–110 it will rapidly fall to the healthy standard, or even below it, when the period of excitement closes.

The Blood.—Accurate observations, on the physical characters and chemical properties of the blood in Yellow Fever, are much wanting to improve our knowledge of the pathology of the disease.

The following facts have been observed from time to time by various authorities:—

1. Blood of a bright scarlet colour has sometimes been drawn at the very onset of the malady.
2. It gradually acquires a darker hue as the disease advances.
3. As observed on both sides of the heart, arterial and venous blood exhibit no appreciable difference.
4. It may present the appearance of being composed of two differently coloured fluids.
5. It may assume a brighter colour as it flows from the arm, or on exposure to the air.
6. It may be of the consistence of molasses, or a thin fluid from

the commencement, though this is more commonly observed in the third stage, or only after death.

7. In many instances the blood remains without coagulation, or is very slow in the process.

8. The crassamentum appears as if it were undergoing solution at the base.

9. The serum varies in colour from a whitish appearance, through yellow and orange, to a red, which has been compared to the tint of water in which meat had been washed.

10. The amount of serum with respect to the clot is smaller than in other cases, and at the commencement than at the close of the malady.

11. In all cases in which yellowness of the skin presents itself, the serum is also found to be yellow from solution of the colouring matters, and the blood globules, broken up from their nummular arrangement, are precipitated to the bottom.

12. The morbid discolourations of the blood, of even healthy persons residing within the range of infection, shows the agreement of Yellow Fever in this particular with what has been observed in the case of other zymotic diseases.

13. Dr. Blair has shown that the dark grumous character of the blood in hæmorrhages supports no necessary assumption that the blood within the vessels is of the same nature.

14. Persons who have had much to do with bleeding in Yellow Fever affirm that the odour of the blood, like that of the skin,[1] is quite characteristic of the disease, differing appreciably from the odour emitted by the blood in other fevers.

15. Dr. Davy has demonstrated the acidity of the blood in Yellow Fever, while alkalinity of that fluid is known to exist in other zymotic diseases.

16. Chassaniol has detected a larger amount of urea in the blood, more particularly in that of the third stage, when the urinary secretion has been more or less suppressed. But Professor Rogers, who proved an excess of salts, contrary to the views of Stevens, was unable to obtain urea in any of the stages. His specimens, however, were believed to have been in a semi-decomposed state. Chassaniol, with great reason, refers the more important symptoms of the adynamic stage to the presence of urea in the circulation.

17. As in other diseases arising from a specific poison, the proportion of fibrine in the blood is much below the normal standard in Yellow Fever; and it is probable that whenever buffing and cupping of the blood occurs in this disease the condition is due to the co-existence of some inflammatory complication.

18. The destruction of the cell wall of the blood corpuscles, said to have been observed by M. de Bienperthuy, is, in the writer's opinion, a doubtful appearance, and certainly not borne out by the researches of Professor Leidy and Dr. Davy.

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[1] See Barruel's remarkable experiments, in which even sex has been determined by the odour of the blood, so closely resembling that of the cutaneous secretions.

Tendency to Hæmorrhage.—As a hæmorrhagic tendency is often developed, more particularly in the third stage of Yellow Fever, it may be well to notice some of its leading features in this place.

Active congestion usually precedes the extravasation of blood in the parts affected, and discharges of this kind have happened from all the outlets of the body without exception. But the special seat of the hæmorrhage, and the amount of it, will vary with the type, or perhaps with the period or locality, of the epidemic.

Hæmorrhagic oozing from the skin, without abrasion, is known at Martinique under the appellation of "sueur de sang." Epistaxis, however, is much more common, and usually happens in the third stage, though it has been known at the close of the first.

Blood may exude from the inner canthus of the eyes, and from the auditory passages, but these hæmorrhages are of rare occurrence. Bleeding from the cavity of the mouth, tongue, gums, and lips is usual in every epidemic; and though the blood may be at first well-coloured, when it mixes up with the secretions of the mouth its character becomes altered, and it assumes a dirty madder-brown hue, coating tongue and teeth.

The menstrual discharge, when not profuse at this period, appears to exert a salutary influence on the disease; but more active coincident hæmorrhage, as in parturition or abortion, are eminently perilous.

Hæmatemesis scarcely ever happens without the passage of blood by stool, which is often critical in its nature.

Ecchymotic abscesses occurring in the neck produce distressing symptoms from their pressure on important parts; and if it be found necessary to relieve them, the writer would recommend a valvular opening. As the patients advance towards recovery, the bloody, ichorous discharge will gradually assume a more purulent character, until finally laudable pus is formed, and the cure goes on in the usual way.

White and Black Vomit.—Up to the commencement of the second stage of the disease, namely, on the second, third, fourth, or fifth day, the ejections from the stomach have been observed to be alkaline; subsequently to which they gradually give an acid reaction, and the discharge of a limpid, ropy, more or less transparent or opalescent, fluid, known as "white vomit," precedes the black, and passes insensibly into it.

Dr. Davy was of opinion, reasoning from analogy, that the white, or precursory, vomit would be found to be of a serous nature, and contain albumen; but if it "remains clear on the application of heat and nitric acid," as stated by Dr. Aitken, the presence of albumen is so far negatived.

As conciseness of matter must be our object in the present article, without entering minutely into the merits of the prolix reasoning of pathologists, in favour of the bilious or of the sanguineous theory of black vomit, the following summary may be given as a fair conclusion from the facts adduced.

When hæmorrhagic oozing begins to take place from the congested lining membrane of the stomach, the blood *ab initio* intermixes with the existing and concomitant acescent secretions of the organ. The blood globules aggregated in masses lose their coloured contents by exosmosis, while they become distended with a thinner fluid, and blend together with a common connecting and finely granular substance, composed of coagulated albumen and the liberated hæmatoglobulin. Dark madder-brown flocculi thus result, floating in a more or less homogeneously-tinted or colourless liquid, and this is probably the simplest definition of black vomit.

Free hydrochloric acid is the grand source of the acidity, tested with litmus and brown turmeric papers.

Nitrate of silver throws down a white precipitate, which is redissolved by ammonia, though not by nitric acid.

Liquor potassæ being added to black vomit, in larger quantity than that required to neutralize its acidity, the flocculent sediment becomes dissolved, with the disengagement of ammonia. It effervesces with alkaline carbonates, and Dr. Blair neutralized ℥xij with ʒi of carbonate of potash.

At the request of Dr. La Roche, Professor Rogers examined several specimens of black vomit with a specific gravity, ranging between 1·003 and 1·016, with the following results :—

Albumen
Sulphuric acid in a state of combination
Chlorine
Alkaline bases
Earthy phosphates
Iron
Hydrochloric acid in a free state.

In his communication to Dr. La Roche, he says :—" These substances, although not the sole ingredients of the blood, are yet, all of them, with the exception of free hydrochloric acid, constituents of that liquid—a fact which, taken in connexion with other characters, and especially the microscopic appearance of the liquids, gives strong evidence that they contain much altered blood ; indeed, the presence of several of the substances enumerated, as albumen, iron, and sulphuric acid, seems not to admit of any other explanation, since it could scarcely be possible that in that stage of the disease they were the results of any food remaining in the stomach."

On submitting black vomit to microscopic examination all the changes above noticed with regard to the blood may be distinctly traced out, but no importance can be attached to the presence of torulæ or other humble vegetable organisms, which are by no means constant. The mention of fatty globules, epithelium (except perhaps that of the tubular glands), starch corpuscles, chicken muscle, and other matters introduced from without, only add scientific jargon to the plain facts of the case, and tend to becloud them.

Mr. Wharton Jones states, that in the contents of the hepatic ducts of man and the sheep, extracted by means of a forceps and without injuring the organ, hepatic cells may be detected. This fact is a difficult problem for the microscopic anatomist to solve, but it sinks into insignificance when he is told that Dr. Blair has not only found the glandular cells, but what he conceived to be "the radical secreting ducts of the liver," in black vomit.

The Urine.—The urinary secretion is generally diminished in quantity from the onset of the malady, and in many instances becomes almost or altogether suppressed, when the symptoms of uræmic poisoning may be developed. Dr. Blair, who was an excellent observer, affirms that it is always acid in the first stage of the disease, and gives an alkaline reaction during convalescence, or when bile is present in large quantity.

Albumen makes its appearance generally on the second or third day, and it may be detected in all severe cases by its appropriate tests.

The colour of the urine, in passing from its natural standard, changes to a bright yellow, a dirty orange, a greenish brown, and olivaceous black, or to a more or less positive red, from the presence of blood.[1]

The ordinary post-mortem appearance of the kidney, to say nothing of its minute anatomy, precludes the idea of the physical detachment of its capillary vessels, or even the Malpighian tufts, and their escape through the tubular system, so as to be commonly witnessed in the urine, as stated by Dr. Blair. Dr. Aitken observes, that this admits of doubt, and that it is not borne out by Blair's own specimens preserved in the cabinet of microscopic preparations of the Army Medical School at Netley. Tube casts, fatty cells, free fat, and blood discs, full and emptied of their contents, with such appearances as often characterise the urine in common jaundice, were the almost invariable objects observed by the writer. There was perhaps a large amount of torulæ and allied forms; but, as in the case of the black vomit, notice of them is of little importance.

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[1] "On chemical analysis," writes Dr. La Roche, "urine taken from the bladder after death has been found greatly deficient of urea. In one case 200 grammes (51 drachms) of the fluid contained but one gramme and $\frac{90}{100}$ (about 16 grains) of urea, no uric acid, and 0·45 per cent. of albumen. In another case, 15 grammes ($\frac{1}{2}$ oz.) of the fluid, obtained in the same way, gave 0·08 of urea, 2·50 of albumen, and no uric acid. In several other experiments, conducted in the same way, the results were similar. Finally, in one case, the urine was examined in the first stage of the disease, and a short time after the death of the patient. In the first the fluid was found to contain, in 100 parts :—

Water	76·08
Urea	2·64
Albumen	0·40
Uric acid	0·08
Earthy phosphates, sulphates, alkaline phosphates, and chlorates	0·80
	80·00

"After death, 20 grammes gave more traces of urea, 0·50 of albumen, and no uric acid."

Morbid Anatomy.—*External Appearances.*—Should the individual die soon after the invasion of the complaint, the bulk of the body may be a little increased on account of a slight puffiness of the skin; but, drained by hæmorrhages and with no repair of the vital fluid, emaciation must happen in more protracted cases.

The eye loses the suffused redness it previously exhibited, and the yellowness of the skin becomes more intense; indeed, it sometimes happens that the skin, which could scarcely be called yellow before, acquires that hue after death, and presents a remarkable contrast with the rich purple blotches and marblings in the more depending parts of the body.

The extremities of the fingers and toes and the tips of the ears are also darkened with stagnant blood.

The tissues in general appear to become abnormally friable, even though examined very soon after death: this is particularly the case with the muscular system, including the heart itself. The flesh is also of a dingy colour, watery, and sodden. It is, however, much paler and softer in those who have suffered much wasting. The connective tissue, moreover, is in general loose and sanguinolent—a condition also observable in malarious Yellow Fever, which is admitted to be a distinct type of disease. The areolar and adipose tissues often exhibit a yellowness similar to that of the skin.

Nervous System.—The brain has been very closely examined in numerous cases, with nearly the same result, namely, that no pathological condition in the slightest degree noteworthy has been detected, even including those cases in which cerebral symptoms, such as active delirium or profound coma, existed before death. (Gillkrest.)

The spinal marrow on the contrary usually exhibits a congested state of the vessels, more especially in the lumbar region, where also the arachnoid membrane has been supposed to be in a state of inflammation.

Effusion of blood has been found in the canal, but we cannot be certain of all that has been said about the existence of actual inflammation. The ganglionic system has also been examined, and with apparently still more definite results. Thus, the semi-lunar ganglia, solar, cæliac, hepatic, and neighbouring plexuses, as well as the connective tissue investing them, have been stated, on the authority of Dr. Cartwright and others, to be uniformly in a condition indicative of inflammation. Nevertheless, excellent observers are not wanting who have never been able to discover any decidedly abnormal state of this system.

Respiratory Organs.—The lungs have been carefully scrutinized in epidemics occurring in different countries within the range of Yellow Fever, and the only important particular, not due to other complications, appeared to be small sanguinolent effusions into the connective tissue of the organs themselves, and that immediately beneath the pleuræ.

Circulatory System.—Usually no very abnormal change is observable in the heart, if we except a dusky and flabby appearance which it often presents in common with the muscular system generally. Professor Riddell and others have laid much stress upon the pretty constant molecular degeneration occurring in the muscular fibres of the heart, with a more or less complete obliteration of their transverse striæ; but it is very probable that this condition, which is perhaps more usual than physicians in general imagine, would be detected in the same subjects had they died of any other malady.

In the pericardium, effusions take place, with or without marks of inflammation, and the fluid may be simply serous, purulent, or sanguineous; and in one instance, thus described by Dr. Bache, the pericardium contained four ounces of a very turbid greenish-brown fluid, resembling black vomit. On pouring this into a bottle and allowing it to stand a short time, it separated into two portions: that at the bottom was of a white or pale yellow colour, while the rest remained without change. An examination by the microscope proved the brownish fluid to be composed of altered blood corpuscles, with less of the granular amorphous matter than is usually found in genuine black vomit. The whitish fluid was pus. The heart itself was stained of a dark colour at its base, and the pericardium was minutely injected in points. (La Roche.)

Transparent amber-looking clots have been frequently detected in the cavities of the heart, particularly on the right side. In fifty-four post mortems made by Dr. Pennell, at Rio Janeiro, clots of the same kind were present.

The endocardium commonly presents no signs of inflammation.

The Stomach.—As might be expected, morbid appearances are more constant in the stomach than in any other organ, so intimately associated as it is with the most serious symptoms of the disease. The effusion of the colouring matter of the blood into the subserous connective tissue may give it a yellowish appearance, but, more unusually, no abnormal change presents itself externally.

A certain amount of the matter of black vomit is generally found in the cavity of the stomach, in some stage of conversion, from actual blood to a dark grumous fluid, like that ejected by the patient while yet alive. It would seem as though the mucous membrane of the stomach were called upon to compensate for the defective secreting and eliminating power of the kidneys; and in those cases where little or no black vomit was found in the stomach after death, the lining membrane presented a thickened appearance with a muco-sanguineous coating.

Sometimes the interior of the stomach presents little or no trace of congestion or inflammation where the mucous membrane has been cautiously washed; while at others the little orifices of the tubular glands are seen to be filled up with a dark brown matter, that may be dislodged with the point of a needle. This appearance has been ignorantly spoken of as the open orifices of the hypothetical vessels, known to older anatomists as the "exhalents," in the act of elimination.

There appears to be no relation whatever between the amount of congestion observed in the mucous coat and the quantity of matter thrown off from it into the cavity of the stomach. Thus the hæmorrhagic oozing may have emptied the capillaries, in some instances, while they still remained congested by the "vis a tergo" filling up the loss in others.

The mucous membrane often becomes mammillated, thickened, the longitudinal folds enlarged, and it is more easily stripped off than usual; but veritable proofs of inflammation, though decidedly present in some cases, are far from being universal in this disease. The tints of congestion, ranging from rose red to claret purple, and dingy grey passing into shades of green and greenish yellow, impart an iridescent appearance to the mucous membrane.

The Intestines present much the same external appearance as the stomach, but the glandular organs, Peyer's patches in particular, are not so uniformly in a morbid state as they are in other fevers. Indeed, the duodenum and upper part of jejunum are often more seriously affected than the ileum.

The Liver is said to become soft and friable in those who die within forty-eight hours, and a serous discharge may take the place of natural bile.

The gall-bladder is usually found empty, or with a small quantity of tarry-looking bile in its cavity.

The *capsule* and fat surrounding the *kidney* have a yellowish appearance, but the organ itself, though prone to fatty change, even during the short period of the disease, is yet not so decidedly involved in it as the liver. Indeed, under ordinary circumstances, an amount of congestion in the liver, only producing the slightest biliary derangement, would be a very serious occurrence in the kidney; and though the latter is a much smaller organ, it would appear to occupy a longer time in passing on to a state of fatty degeneration. Little livid spots and ecchymoses are sometimes found in the infundibulum and pelvis, and pus has been found in the pelvis and ureter. Dr. Pennell notices, besides the manifest congestion of the kidney, that the papillæ yielded upon pressure a glutinous tenacious exudation sufficient to interfere mechanically with the escape of the urine. This, however, the writer is disposed to think was in great part composed of the forcibly extruded epithelial lining of the little tubes in a manner well known to microscopic anatomists.

The *pancreas* and *spleen* present no constant pathological change worth noticing, and the same may be said of the *urinary bladder*, if we except the possible occurrence of a dark grumous fluid, closely resembling black vomit, recorded by one or two observers.

PROGNOSIS.—It is a difficult matter to form a reliable prognosis in almost any stage or variety of Yellow Fever, for the very cases that would strike the physician as affording most promise of speedy recovery may prove to him, by a rapidly fatal issue, how uncertain his judgment must be. Still, there are symptoms or circumstances which experience has shown to be usually of good import, and others again which augur badly or point to an almost immediate dissolution.

Favourable Signs.—If the pulse approximates without reaching 110 in the febrile stage;[1] if it preserves an even and normal force and frequency when the third stage sets in; if the urine be in good quantity and exhibits no disorganization of the kidney, under the microscope—the presence of albumen in small amount is to be regarded more as an usual than an unfavourable symptom. If epigastric tenderness and irritability subside with the febrile state, a good hope of the case may be entertained.

Unfavourable Signs.—Dr. Jackson gives the following as indicative of danger, founded upon his experience of the disease, occurring on the south coast of Spain:—

1. A sudden invasion by the fever, with intense pain of the head and eyeballs, accompanied by sickness and vomiting.
2. The fever being ushered in by a fit, convulsions, or apoplectic stupor, or outrageous delirium.
3. A torpid, heavy, or statue-like aspect of countenance gave strong suspicion of danger.
4. A dry, rough, milk-white, or swollen and red tongue indicated danger.
5. Distress and anguish at stomach, with pain at the epigastrium, forcible eructations, or explosions of flatus from the stomach, gave impression of much danger.
6. Obscure hiccough marked danger.
7. A ghastly appearance, with a faint nauseous odour from the body, indicated extreme danger.
8. Yellowness of the skin, with turgid veins on the conjunctivæ in the latter stage, "always decisive of a fatal issue."
9. Torpor of the skin—to such an extent as to be insensible to the stimulation of blisters and sinapisms—is ranked among dangerous signs.
10. Extreme dampness or extreme dryness of the skin indicates great danger.
11. Petechiæ are suspicious: streaks, or patches, of livid green, or violet colour, are almost certain indications of approaching death.
12. Vomiting of black matter, like the grounds of coffee, is reported a sign of the highest danger; [but]
13. Vomiting of bitter bile, whether green or yellow, even with straining and severe retching, affords a sign of comparative safety.
14. Black watery stools, with shreds, "are of the worst prognostics" (as quoted by Martin).

To the above may be added:—

1. A very weak pulse on the invasion of the disease.
2. A pulse much exceeding 110 in the febrile stage.
3. Sudden and excessive lumbar pain, or rachalgia.

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[1] The frequency of the pulse as a rule being less in Yellow Fever than in others.

4. Countenance swollen, tense, and bloated, or on the contrary much pinched up, or terror-stricken.

5. Fiery redness, with prominence of the eyes, or on the other hand a pearly whiteness of the conjunctivæ.

6. Widely dilated pupils, indicative of cerebral complication.

7. Suppression of urine, or deposits indicating a serious condition of the kidney.

THERAPEUTICS.—*Treatment.*—There can be little doubt that whatever is to be done in Yellow Fever should be done quickly, and the earlier a clear diagnosis is formed the better, if any hope may be reposed in medicine.

We have it on the authority of physicians of experience, that the disease has been cut short by the timely administration of remedies that have been tried and found wanting at a later period. Dealing with the subject in a purely philosophical light, the evidence before us is not as conclusive as it could be wished; on this point, and "in the present state of our knowledge, we can be only guided by the common principles of medicine; for it is plain that a specific mode of cure cannot be suggested until the nature of the specific cause is known. We know also that the disease manifests itself with various degrees of intensity in different cases, and how much of any happy recovery depends upon the stamina of the system, or upon the means employed, is, above all things, difficult to determine—a fact which should never be forgotten by those who may be zealously inclined to advocate their own suggestions." Even a brief notice of the various modes of practice adopted by medical men from time to time, in the treatment of this disease, would occupy more space than would be of advantage in the present article; but we shall pass the more important remedial agents in review.

The *pediluvium* on the first invasion of the malady is now very largely employed by American practitioners, and the indication is further carried out by the use of warm drinks to excite diaphoresis and a genial warmth. This may be followed by a brisk purgative, composed of calomel, gr. vi.–x., jalap, gr. x., and ginger, gr. iii., in the bolus form, which will be found the most convenient and certain mode of administering medicines of this kind on an extended scale.

A dose of calomel, exceeding ten grains, would appear to be rather experimental or empirical than based upon a sound physiology. No one has been able to advance a therapeutical principle to warrant it, in opposition to the evil effects so often known to follow in its wake. The same also may be said of large doses of quinine, which, in Yellow Fever at least, only tend to impede secretion and derange the circulation within the head. The employment of this medicine at all is more suitable to the convalescent than to the patient more immediately under the influence of the disease. It is perhaps more useful in such febrile states as exhibit a periodicity in their recurrence. Should it be thought expedient to prescribe quinine in Yellow Fever, small doses, frequently repeated, are to be preferred, so that any resulting prejudicial effect may be observed in good time, and the medicine simply omitted without doing much mischief.

As the bowels are commonly sluggish in Yellow Fever, an enema may be necessary after the first dose above suggested; in which case one of turpentine, as recommended by Drs. Copland and Smith, will prove to be the most beneficial, as it in general brings away feculent and normal-looking stools. Turpentine, in drachm doses, by the mouth, is advocated by the same authorities, but of its use in this way the writer has had no experience.

The most must be made of the time now remaining until retching and vomiting set in. Acetate of ammonia, nitrate of potash, nitrous æther, and the tincture of squills and henbane, may be combined and administered in periodical doses, with lime juice for drink. In this way excessive febrile action will be moderated, the action of the kidney and the skin preserved, and even if the reaction is defective no further depressing influence can be exerted.

Of all the symptoms of Yellow Fever, the most distressing, to both patient and the physician, is irritability of the stomach. It is so constantly present, and so often uncontrollable, that the knowledge of every available means of checking it is of the greatest importance.

In the epidemic of Yellow Fever on board H.M.S. *Icarus*, in the West Indies, a few drops of chloroform prepared the stomach for the reception and retention of food, "but the dose should be repeated a short time before food is again taken, as the effect of chloroform is transitory."

Creosote and hydrocyanic acid have been used with the same intention, but the valuable suggestion of chlorodyne, made by Dr. Aitken, would bid fair to supersede every other, if we may except that of lime water, in this connexion. Lime water was used with great benefit on the *Icarus*, on the recommendation merely of its known therapeutic properties in the practice of medicine. But it had long previously been employed by the American physicians, and with a higher object, namely, the correction of acidity in the stomach, due to the presence of free hydrochloric acid. Dr. Hosack used lime water most successfully in combination with milk, and with porter when the milk could not be retained. More stress ought to be laid upon the importance of this agent by English writers. A chalk mixture has also been resorted to with the same intention.

Professor Frost, of Charleston, strongly advocates the chlorate of potash; and it is richly worth extensive trial, from its known valuable properties in adynamic states generally, oxygenating the blood, and aiding in elimination by its action on the skin and kidneys. It is fair, however, to state that some cases, in which it was tried, were not very satisfactory.

Tannic acid, besides the astringency which it exerts upon the smaller vessels, is reputed to have the property of diminishing the irritability of the stomach.

Pepsine, in regulated doses, so as to assist an organ incapable of discharging its own functions efficiently, requires further trial. In connexion with pepsine, and the internal use of ice in the intervals of its exhibition, the essence of beef should be unremittingly supplied, and suited by dilution and seasoning to the nature of the case. This will be the time also for stimulants—our great object being to obviate the tendency to death, and to sustain the vital power in its struggle with the foe. Champagne, in particular, will be beneficial, when it can be obtained. The ward-room cook of H.M.S. *Icarus* had very nearly succumbed at this crisis, but he rallied immediately on the administration of a stout glass of rum and water, and recovered steadily.

The Yellow Fever poison is evidently of an intoxicating kind, and a marked difference is apparent between the mild and irritable delirium produced by it, when thrown out in contact with the brain substance, and the comatose state brought about by those principles, urea in particular, that accumulate in the circulation in consequence of the ineffective action of the kidneys and liver. When the semi-comatose patient is aroused, he may be made to answer rationally; but in the delirium, which is characterised by vigilance and irritability, this can scarcely be expected, and even a coherent reply may be in league, as it were, with the mental derangement. Morphia certainly has the effect of allaying this excitable state and procuring sleep. Here, again, Dr. Aitken suggests chlorodyne, as the administration of "opium in any form," on good therapeutic grounds, is objectionable.

VARIETIES, AND THEIR CLASSIFICATION.—Notwithstanding all that has been written on the subject of Yellow Fever, the gist of nearly every attempted classification of its varieties amounts to little more than a grouping of them in accordance with the several degrees of comparison. Indeed, the phases presented by different epidemics, and by the individual cases in the same epidemic, with no possibility of the interpolation of other complaints to be confounded with it, would give systematists more than they could easily accomplish to define and arrange intelligibly. The types, therefore, are exceedingly variable, and little practical benefit can accrue from their nice discrimination; but, when they do not transgress broad boundary lines, they may afford a more precise knowledge of the symptoms and pathology of the disease as a whole. It is notorious that a classification, founded upon the experience of one epidemic, may be quite inapplicable to another; consequently, whoever has to do with one, naturally makes a classification for himself. On this account, systems have become so numerous, that we can only give place to a notice of one of the best of them, namely, that put forward by Dr. La Roche of Philadelphia, whose masterly work on Yellow Fever, with a bibliography of sixty-one pages, has afforded the writer great assistance in the composition of this article.

According to Dr. La Roche, Yellow Fever (as a genus) is divisible into two species, viz. 1, the Inflammatory; and 2, the Congestive; though these are often connected by cases in every conceivable degree of transition from one to the other.

1. The inflammatory species appears under three grades, viz. (*a*) the Intense; (*b*) the Mild; and (*c*) the Ephemeral.

From all that has been previously said in the symptomatology, the character of these grades may be easily conceived. In the *first*, the fever is active and short, and death commonly happens before the accession of black vomit; in the *second*, the fever is more protracted, and may even exhibit partial remissions before the final stage sets in; while the *third*, as its name implies, is of short duration, and easily amenable to treatment.

2. The Congestive species is marked by the passage of the disease "as it were directly from the first sign of indisposition to the last stage, without going through that of reaction." Of this species there are four grades: viz. (*a*) the Aggravated; (*b*) the Adynamic or Typhoid; (*c*) the Walking; and (*d*) the Apoplectic.

(*a*) The Aggravated grade is attended from the commencement with considerable prostration, giddiness, stupor, and loss of memory, delirium, or coma. The tongue is natural, or with white patches and red tip and edges. The countenance is livid and apathetic, and the skin yellow or bronzed. Hæmorrhage occurs from one or more of the natural outlets, and the patient keeps up a low monotonous wailing. In some cases the pulse is nearly natural, the tongue clean, and the stomach calm; but these are attended with excessive restlessness, anxiety and distress, soon followed by black vomit and fatal collapse.

(*b*) The Adynamic or Typhoid grade occurs in persons deficient in vital power, ushered in with chills, followed by burning heat, partially distributed over the body, viz. principally on the under parts of the arms and inner surface of the thighs. The pulse is small and weak. The skin assumes an olive hue, and is covered with petechiæ or vibices. Hæmorrhages are common from the natural outlets, or into the connective tissue beneath the skin, or amongst the muscles.

(*c*) The Walking grade.—Here the functions of organic life appear to be at first alone implicated, those of animal life remaining unaffected. The patient, though sometimes in bed, is found more frequently walking about his room. He only feels weak, but his eye is watery, and his countenance dull and listless; his pulse grows fainter and fainter, until at last he is overtaken by black vomit and death speedily ensues.[1]

(*d*) The Apoplectic grade.—The patient is more or less suddenly struck down with stupor or coma, and death, preceded by convulsions, soon follows. The pulse is always weak, and finally becomes faltering. The skin is cold and clammy, or sometimes dry and flabby. "In the meantime the patient lies as if stunned, with dilated pupils and an expression of gloom upon the countenance. From this unpromising state an effort at reaction occasionally takes place, but this scarcely ever leads to a successful result. More generally, the patient becomes perfectly comatose, the eyes assume a glassy appearance, the pulse fades away, involuntary discharges and profuse hæmorrhages supervene, and death soon ensues."

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[1] Several well-marked cases of this grade occurred on board H.M.S. *Icarus.*

Some few years ago[1] the writer surmised the possibility of the extension of the range of Yellow Fever to our own shores, all favourable conditions being fulfilled, and this has been realized in the late epidemic of Yellow Fever at Swansea, reported in a very able manner by Dr. Buchanan. The circumstances are briefly the following :—

On the 9th September, 1865, the *Hecla* barque, laden with copper ore, returned from Cuba to Swansea, with one case of Yellow Fever on board, three having proved fatal on the voyage home.

The remaining case (James Saunders) and two convalescents were sent on shore. Soon, also, the crew had left the ship and distributed themselves over the town, and two passengers with their luggage were landed. Moreover, a good many people boarded the vessel as she entered the dock.

An outbreak of Yellow Fever thus originated ashore, and in a well-drawn-up table of the cases Dr. Buchanan shows their invariable connexion with the source of the malady, directly or indirectly, adding further proof of its infectious nature, should such be required.

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[1] Health of the Navy for 1860.

NOTE.—As bearing upon the question of infection, the following important quotation is from the remarks of Deputy Inspector-General R. D. Mason of Port Royal Hospital, recorded in the Statistical Report of the Health of the Navy for 1866 :—

"During the Christmas quarter nine cases of Yellow Fever have been received into hospital, of which eight have terminated fatally. The first case received was that of the commanding officer of the gunboat *Nettle*. The *Nettle* had been employed between the 1st of October and 25th of November at Morant Bay and Port Morant, spending a week alternately at each place. At Morant Bay this officer resided on shore, passing scarcely any time on board. The servant died of Yellow Fever at Morant Bay on the 22d of November after about four days' illness ; and a staff assistant-surgeon, who lived in the same house and who attended him, was subsequently attacked with the same disease and died on the 10th of December.

"Up to the time that the *Nettle* left Morant Bay no other cases were known to have occurred, and there had been no unusual amount of sickness, nor any cases of Yellow Fever, as far as I have been able to ascertain.

"The surgeon of the *Cadmus* states, however, that as far back as the month of July, it was reported that a merchant-ship from St. Thomas, then lying at Morant Bay, or one of the eastern ports of the island, had cases of Yellow Fever on board : cause and effect are also shown in the fact that the chaplain of the *Cadmus* had on several occasions visited the commander of the *Nettle* during his illness, and that the surgeon was at his bedside about an hour before his death, and that the *Cadmus*, previously a healthy ship, had subsequently sent eleven cases of Yellow Fever to hospital."

Circumstances like these may appear very unimportant at the time of their occurrence, but it may be safely affirmed that such are always traceable in connexion with the communication and spread of Yellow Fever.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

BY J. NETTEN RADCLIFFE.

DEFINITION.—An acute, epidemic disease, characterised by profound disturbance of the central nervous system; indicated, at the outset, chiefly by shivering, intense headache or vertigo, or both, and persistent vomiting: subsequently by delirium, often violent, alternating with somnolence, or with a state of apathy or stupor; an acutely painful condition with spasm—sometimes tetanoid—of certain groups of muscles, especially the posterior muscles of the neck, occasioning retraction of the head; and an increased sensitiveness of the surface of the body. Throughout the disease, there is marked depression of the vital powers; not unfrequently collapse: and in its course an eruption of vesicles, petechiæ, or purpuric spots, or mottling of the skin, is apt to occur. If the disease tend to recovery, the symptoms gradually subside without any critical phenomena, and convalescence is protracted: if to a fatal termination, death is almost invariably preceded by coma. After death, the enveloping membranes of the brain and spinal cord are found in a morbid state, of which the most notable signs are engorgement of the blood-vessels, usually excessive, and an effusion of sero-purulent matter into the meshes of the pia mater, and beneath the arachnoid.

SYNONYMS.—(a) *Technical*:—Cerebro-spinal fever[1] (*Royal College of Physicians*); cerebro-spinal arachnitis; typhus syncopalis; tifo apoplettico tetanico; typhus cérébro-spinal (*Boudin*); cerebral typhus; epidemic meningitis (*Stillé*, U.S.); petechial fever (*G. B. Wood*, U.S.); fever with cerebro-spinal meningitis (*S. Gordon*); malignant purpuric fever (*W. Stokes*); malignant purple fever; nervo-purpuric fever (*Mapother*); malignant purpuræ (*M'Swinney*); pestilential purpuræ

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[1] Since the completion of this article the Royal College of Physicians, in its "Nomenclature of Disease," has adopted the following designation and definition of this malady: "*Cerebro-spinal Fever*. A malignant epidemic fever, attended by painful contraction of the muscles of the neck, and retraction of the head. In certain epidemics it is frequently accompanied by a profuse purpuric eruption, and occasionally by secondary effusions into certain joints. Lesions of the brain and spinal cord and their membranes are found on dissection." It is, however, determined to retain the name by which this affection was described in the first edition of this System of Medicine, and for the simple reason that such name, while sufficient for the purpose of recognition, conveys no opinion as to the nature of the disease.—EDITOR.

(*Banks*); febris nigra (*R. D. Lyons*).—(b) *Popular*:—Spotted fever (*New England*); cold plague (*Southern States*, U.S.); Kolik, Nackenstarre, Genickkrampf (*Germany*); Nacksjuka, Dragsjuka (*Sweden*).

DESCRIPTION OF THE DISEASE.—I. *General Symptoms*:—Epidemic cerebro-spinal meningitis is observed in three principal forms: A.—*Simple*, in which the symptoms indicative of disorder of the nervous centres predominate throughout the whole course of the disease; B.—*Fulminant*, in which the depressed state of the vital powers, with profound blood-change—as shown by hæmorrhage of various forms into the cutis—characterises the disease; and C.—*Purpuric*, in which the cerebro-spinal symptoms, and the symptoms which mark blood-change (*petechiæ*, *purpuræ*, *vibices*, &c.), and flagging of the vital powers, occur together. The proportion in which the three forms of the disease are manifested varies considerably in different epidemics. In every outbreak cases are observed which link, by insensible gradations, one form with another; while in other, and rarer cases, the characteristic symptoms of the three forms are merged together. Continental and American writers have described an *abortive* form of the disease, the term being given (*a*) to certain anomalous symptoms observed in communities among which the disease is active: and (*b*) to sundry symptoms characteristic of the malady, but of transitory duration: such as severe cephalalgia, a sense of dragging at the back of the neck, or actual slight retraction of the head; cardialgia, enteralgia; —these symptoms often ending contemporaneously with the appearance of profuse perspiration, or epistaxis.

(A.) *Simple Epidemic Cerebro-spinal Meningitis.*—In the majority of the cases before the onset of the disease the patient suffers from more or less indisposition. There are discomfort in the head, neuralgic pains in the back, the principal groups of muscles, and the abdomen; failure of the appetite, indifference to exertion, perhaps also slight shiverings, and a quasi-febrile state. These indications of disordered innervation may persist from three to seven days, or may be manifested only during a few hours, before the confirmed malady fully declares itself. But in numerous cases the onset of the disease is sudden and characteristic. In both classes the accession of the malady is declared by similar well-marked signs. Acute shivering is followed or accompanied by severe, commonly intolerable, headache, or vertigo, or both; and after a short interval, or contemporaneously, profuse and irrepressible vomiting takes place, rarely preceded by nausea. Or vomiting may be the initiatory symptom, the shivering, headache, or vertigo following quickly after. The intensity of the symptoms marking the onset of the disease is remarkable and characteristic. The sickness is often, and from the outset accompanied by severe abdominal pain, apparently neuralgic; and not unfrequently this pain precedes the disorder of the stomach, as the cephalalgia precedes mental confusion. In like manner, the shivering ushers in, or is accompanied by, an acutely painful state of the muscles, more or less general, the forerunner of spasm. Cephalalgia and delirium, abdominal neuralgia and vomiting, and myalgia and spasm are the principal morbid factors of simple epidemic cerebro-spinal meningitis. They distinguish the malady, and the varying prominence with which they occur in different outbreaks gives rise to many diversities in the grouping of symptoms during the progress of the disease. The onward course of the disorder is usually rapid. The headache continues, often without a lull; vertigo occurs frequently; and after the lapse of a very brief period, measured usually by a few hours, the mind becomes confused, and, in some cases, a state of restlessness supervenes not unlike that observed in delirium tremens. The mental confusion assumes the form of muttering delirium, with periods of somnolence, often interrupted by cries provoked by the intense cephalalgia, or by the neuralgic pain elsewhere; or the patient falls into a state of apathy or stupor, from which he may be partially roused, but into which he relapses when left undisturbed, the mind acting as in a dream; or there is acute and violent delirium. Contemporaneously with, or immediately prior to, the mental disturbance, the painful state of the muscles increases, certain groups being more manifestly affected than others, especially the posterior muscles of the neck, the muscles of the spinal column, and those of the lower extremities. The pain, often of an acutely neuralgic character, shoots along the spine and limbs, and across the walls of the abdomen. Partly as a voluntary action, partly as a consequence of spasm of the painful muscles, the head is drawn backwards. The retraction thus arising is one of the commonest and most characteristic symptoms of the disease. As the malady advances an actual or apparent tetanoid contraction of other groups of muscles may occur, the trunk most frequently being curved backwards, and the legs bent upon the thighs. At the same time there may be fleeting spasmodic action of some of the muscles of the face, and occasionally of the eyeballs; or in some cases tonic contraction of these muscles, giving rise to the so-called sardonic laugh, or to persistent strabismus. In many cases cutaneous sensibility is much exaggerated, and very frequently a vesicular or roseolar eruption is developed, the former particularly about the lips. The aspect of the patient as the disease advances is dependent upon the degree of pain, the state of delirium or stupor, and extent of spasm which may be present. The countenance is rigid and contracted, the expression of face betokening acute pain; or it is dominated by the delirious fancies; or reflects the mental torpidity; or is distorted by spasm. There is frequently a slight suffusion of the eyes, altogether different from the dusky appearance of typhus; and the face is commonly pale and sunken, seldom and only transitorily flushed and swollen, except when affected more or less extensively by the vesicular eruption. The surface sometimes moist, sometimes dry, rarely gives to the hand a sensation of febrile heat, although the temperature of the body ranges above the normal standard. The pulse from the outset is wanting in firmness, and the indications of defective tone increase as the disease advances. The respiration exhibits no marked disturbance, excepting an increase of rapidity witnessed during accessions of pain and restlessness, and in the advanced stage of the malady the diminution dependent upon failing circulation and innervation. The alimentary canal, apart from the vomiting, which usually ceases as the disease becomes fully developed, presents little indication of disturbance. The tongue is as frequently clean and moist as dry, foul, and discoloured; and the bowels may be either costive or loose, the former, perhaps, more commonly than the latter. In some outbreaks, indeed, costiveness has been marked and almost general, but in others diarrhœa has been prevalent. The renal secretion is rarely much disturbed.

As the malady proceeds, if it tend towards a fatal termination, the spasmodic symptoms increase, the patient becomes comatose, and death may occur either from asphyxia or exhaustion in from ten or twelve hours to seven or eight days. If the disease be prolonged beyond this period, various secondary lesions are apt to occur, especially certain inflammatory states of the eyes and the ears, the mischief in the former organs being shown by ulceration of the cornea, iritis, and sometimes suppuration of the globe; in the latter by less obvious structural changes during life except as indicated by deafness. Or there may be paralysis affecting one-half of the body, or one side of the face, or one of the limbs, or an isolated group of muscles. Or there may be an inflammatory state, with sero-purulent effusion into one or more of the large joints. Or, finally, the patient may fall into a state of marasmus and nervous exhaustion, often protracted and not rarely fatal. If the malady proceed to a favourable termination without any of these sequences, health may be recovered in from three to four weeks. If the progress of the disorder, otherwise favourable, be interrupted by one or other complication, the period of recovery is uncertain and often long postponed.

(B.) *Fulminant Epidemic Cerebro-spinal Meningitis.*—In the fulminant form of the malady the onset is without premonition. The patient suddenly falls into a state of collapse. The surface of the body has often a cyanotic aspect, and is cold and clammy to the touch, or covered with profuse perspiration, the face being not rarely shrunken and livid, and the eyes deep sunk as in the algide stage of cholera. There may be some shivering at intervals, more or less pain of the head, and occasional vomiting, sometimes of a grumous black or coffee-coloured fluid. Drowsiness, if not present at the outset, rapidly supervenes, followed by or concurrently with delirium. Coma, rarely other than the precursor of death, quickly succeeds. In the meantime, purpuric spots show themselves over the surface of the body generally, red or purple and circumscribed in the beginning, but rapidly becoming black, and often extending their margins so as to form irregular inky blotches, or streaks, or great patches; and not unfrequently several of the spots become gangrenous. Sometimes the purpuric spots appear contemporaneously with the collapse at the outset of the attack. The respiration is preternaturally slow, and the pulse (if it has not been absent at the wrist from the beginning) falls with the progress of the disease. The urine is loaded with albumen. Life may be extinguished in less than *five hours*, or it may be prolonged for two or three days. Recovery from this form of epidemic cerebro-spinal meningitis is not unknown, but it is an exceedingly rare event.

(C.) *Purpuric Epidemic Cerebro-spinal Meningitis.*—In the purpuric form of epidemic cerebro-spinal meningitis, the symptoms which distinguish the simple and fulminant forms of the disease occur combined together in various proportions, some cases approximating more or less closely to the latter, others, as is most common, to the former variety of the affection. Thus concurrently with shivering, intense headache, vomiting, rachialgia and retraction of the head, there may be depression of the vital powers approaching collapse, or collapse itself, with the development of petechiæ, purpuræ, vibices, ecchymoses, hæmorrhage from the mucous tracts, delirium, coma, and rapid dissolution. In by far the greater number of cases, however, the disease follows the course of simple epidemic cerebro-spinal meningitis; but within twenty-four hours, or from this period to the fourth day, or still later in the progress of the malady, petechiæ or purpuræ are developed more or less copiously, and occasionally hæmorrhage occurs from the mucous tracts. This phase of epidemic cerebro-spinal meningitis does not appear to be more fatal than the simple form of the disease. It has been observed more commonly in the United States than on the continent of Europe, and it was the principal variety which occurred during the outbreak in Ireland in 1867.

II. *Special Symptoms.*—1. *The Nervous System.*—Headache is almost constant, and it is remarkable for its early and persistent severity. At the outset it is not localised in any particular part of the head. It may be referred to the forehead, the sides, the vertex, or the occiput; or it may be general. Later in the disease, the occiput is, perhaps, most commonly the seat of pain. The intensity of the headache is, as a rule, peculiar. The patients describe the pain as sharp, lancinating, stabbing, plunging, tensive, throbbing, boring, or crushing. It is so intolerable as to elicit groans and cries from the sufferer; often even during delirium or stupor, the exclamations, the contraction of the forehead, and the manner in which the hands are moved towards the head, show that the pain continues. In young children this state closely resembles that which is so significant of tubercular meningitis. The headache may cease when the disease has become fully developed, or, as is probably more common, it may persist throughout the whole course of the malady so long as consciousness remains. Occasionally, indeed, when recovery takes place, it will continue far into the period of convalescence.

Rachialgia is rarely absent. It is sometimes general throughout the spinal region, but more frequently it is limited to the loins, the dorsal region, or, as is most usual, to the posterior part of the neck.

Occasionally the pain radiates from the neck to the extremities and walls of the abdominal and thoracic cavities. In rare cases the pain has commenced at some point of the peripheral nervous system, and spread thence to the back, occurring in paroxysms. This pain has the same character as the cephalalgia, and the words (intolerable, atrocious, tensive, &c.) used to indicate the nature of the latter may be employed also to describe the former. It is augmented by movements, and its chief seat is in the muscles of the spinal column.

The nuchal pain and its consequences constitute one of the most characteristic signs of the disease. Frequently, at the outset of the malady, this pain is preceded by a dragging sensation at the back of the head. As the pain increases in intensity, the head is voluntarily thrown back to relieve all strain upon the exquisitely sensitive muscles. Or, in conjunction with the pain, spasm of the affected muscles occurs, and the head is forcibly drawn backwards. Among the popular terms of the disease, those arising from this symptom (Nackenstarre, Genickkrampf, Nacksjuka, &c.) are very prominent. When the rachialgia is more diffused, and the pain extends also to the limbs, adapted or spasmodic contractions of the trunk and lower extremities are apt to occur. Rachialgia is not present in the fulminant and in severe cases of the purpuric forms of the affection. It is noteworthy that pressure on the spinous processes, during the most acute rachialgia, rarely causes pain.

Enteralgia and other Neuralgic Pains.—Abdominal pain, neuralgic in character, and more or less closely linked to the pain in the course of the spine, is not unfrequent, and it is often closely associated with uncontrollable vomiting. In some epidemics, as in that of 1865 on the Lower Vistula, enteralgia was so common among children seized with cerebro-spinal meningitis that it gave rise to the trivial designation "belly-ache," as one of the popular names of the disease. Neuralgic pains in the limbs, referred to in connexion with rachialgia, are less common than like pains along the course of the spine and in the abdomen.

Increased Sensitiveness of the Surface of the Body has been described as frequent in several outbreaks. During the late prevalence of the disease in the United States (1861-67), cutaneous hyperæsthesia is said to have been a characteristic symptom of the malady in its fully developed state. During the outbreak on the Lower Vistula, an increase of cutaneous sensitiveness was also observed very commonly, but it was not regarded by Dr. Burdon Sanderson as a characteristic symptom, but "a mere consequence or interlude of pain:" being, in fact, an excessive tenderness experienced during intermissions, or after the cessation of pain.

Spasm.—Sufficient care has not always been taken to discriminate between apparent and actual spasm in this disease. Tourdes, in 1843, showed that the retraction of the head and curvature of the spine did not in all cases arise from a spasmodic contraction of the muscles, but that the position was not rarely voluntarily or instinctively assumed by the patient as most conducive to relief of the spinal pain. Dr. Burdon Sanderson confirmed this observation of Tourdes, so far as retraction of the head was concerned, in 1865. In the cases observed by him, in which the head was apparently drawn backwards, it was practicable to extend the seemingly contracted muscles, although the effort gave rise to exquisite pain and instinctive resistance. There was not any tension of the muscles except such as arose from this resistance; no tightness was felt so long as they were at rest. "It was not till the neck was completely extended that the muscles became hard, and even then the hardness was not for a moment comparable to that which is felt in tetanus." The position in bed of the patients observed by Dr. Burdon Sanderson was that which would produce the greatest relaxation of painful groups of muscles. There can be no doubt, however, that spasm is a frequent accompaniment of epidemic cerebro-spinal meningitis. In the clonic form it is witnessed in some cases as transitory contractions of the facial muscles, cramps of the extremities, the convulsive agitation and trembling referred to in the general description as somewhat like what is observed in delirium tremens, very rarely in local convulsion of a single limb, and still more rarely in general convulsion. Tonic spasm of the muscles of the face, jaws (trismus), and gullet, and of the limbs and trunk, may also occur, giving rise to true opisthotonos, emprosthotonos, or general tetanic rigidity of the trunk and limbs.

Paralysis is not of very common occurrence during the progress of epidemic cerebro-spinal meningitis. Hemiplegia has been occasionally noticed, and paralysis more or less complete of one or both extremities, upper and lower, of the muscles of deglutition, of articulation, and of certain other associated groups, the latter chiefly towards the close of the malady. The general paralysis noticed by some writers was usually significant of, and indeed a part of the phenomena of, approaching dissolution.

The *special senses* do not often manifest much change, except as a consequence of certain structural lesions. Increased, sometimes exquisite, sensitiveness of sight and hearing has occasionally been noticed, concurrently with augmented sensitiveness to other external impressions, especially towards the close of the malady, when complete consciousness returns. Amaurosis has also occurred, without apparent change in the ocular apparatus. It may be noted, moreover, of the eye and sight, that occasionally there is strabismus and double-vision. The pupils may be normal in aspect and action, or they may present various changes. They may be dilated or contracted, or one dilated and the other contracted, or they may exhibit curious alternations of contraction and dilatation under the influence of the same degree of light. Both the *eyes* and the *ears* are liable to undergo certain structural lesions, consisting in well-marked inflammatory changes. These commence, in the former organs, sometimes in the cornea, sometimes in the deeper tissues. Most commonly keratitis is set up, ending in opacity or ulceration; and, in the latter case, the iris may become involved. Or, iritis may occur independently, with effusion of lymph or pus, and the consequences thereof (synechia posterior and distortion of the iris are particularly noted). Among more deeply-seated changes may be mentioned opacity of the lens or of the vitreous humour, separation of the retina from the choroid, purulent infiltration, or atrophy of the eyeball. The ear suffers, perhaps, more frequently than the eye. Deafness is probably more common than defects of vision, and it is largely dependent upon inflammatory changes set up in the organ, and particularly affecting the lining membrane of the vestibule and semicircular canals. Occasionally, the external meatus has been affected, and a profuse purulent discharge has flowed from it. These lesions of the organs of sight and hearing may occur either early or late in the course of the disease. The sense of *smell* very rarely suffers. Its loss in one nostril has been recorded in a single case, and this was, perhaps, dependent upon inflammatory changes in the lining membrane of the nose; as purulent discharge from the nostrils has occasionally taken place in other instances.

Vertigo is sometimes observed as an initial symptom of the disease in conjunction with the cephalalgia. Instances are recorded in which the first accession of the disease was marked by severe giddiness, during which the patient either staggered about like a drunken man, or turned round several times, and then fell.

Delirium is rarely absent. It varies much in character, and may occur at any period of the seizure. It may be quiet or violent, transitory or more or less persistent. It sometimes, but rarely, forms one of the symptoms of invasion, when its access is sudden and its character acute. It may supervene with violence after the malady has continued from several hours to two or three days. In the acute form of delirium, the patient is very noisy, and often so violent as to require restraint. Sometimes it happens that paroxysms of furious excitement occur with intervals of placid delirium. Hence the necessity of great watchfulness in the care of these cases. Most commonly the delirium follows closely upon the initiatory symptoms, and is aggravated as the disease advances. At the beginning, the confusion of thought may not be so great but that the patient can be roused so as to answer questions intelligibly. Later, the incoherence becomes much greater, and is usually accompanied with considerable agitation. Much difference is observed, not only in the degree of impairment of the consciousness, but also in the periods of manifestation of the impairment. In some cases, the delirium occurs chiefly during the night; in others, and very commonly, it alternates with periods of somnolence or of quietude. In the more persistent cases there are usually exacerbations. If the disease tend to a fatal ending, the delirium is followed by coma; if to recovery, consciousness is, as a rule, gradually recovered: but, at times, a period of stupor intervenes between the subsidence of the delirium and returning perception. In the slightest cases of the malady the delirium may be transient only, taking place at intervals, and chiefly during the night. In the gravest cases, when death occurs in a few hours, delirium is most commonly present. The duration of the delirium depends entirely upon the nature and duration of the case. Instances are recorded in which furious delirium has occurred for three nights in succession. In other instances a delirious state has persisted more or less continuously for fifteen days.

Stupor and Coma.—In not a few protracted cases, delirium is followed by a prolonged state of stupor, the patient lying completely indifferent to external impressions. In six cases observed by Dr. Burdon Sanderson, in which there had been violent delirium at the outset, this state lasted from one week to five weeks, the mean duration of the several cases being nineteen days. The observer remarks, however, that as four of the cases "emerged from their stupor in a state of complete deafness, there was much difficulty in limiting accurately the period of unconsciousness." Sometimes the state of stupor supervenes without the intervention of violent delirium. *Coma* occurs in nearly all fatal cases, and is, indeed, generally the forerunner of death.

2. *The Digestive System.*—The uncontrollable *vomiting*, which is one of the characteristic initiatory symptoms of the disorder, is an effect of the cerebral mischief. Most frequent at the beginning of the malady, the vomiting diminishes as the disease advances, occasionally increasing during exacerbations. The matter evacuated, after the stomach has been emptied of food, is usually of a greenish or yellowish colour and bitter taste, and is composed largely of bile; more rarely it is viscid and white. Occasionally, in the fulminant and purpuric forms of the malady, a grumous black or coffee-coloured fluid is vomited. In several outbreaks, the vomiting of large quantities of *Ascarides lumbricoides* has been specially noted. The *buccal cavity* and *tongue* do not exhibit any particular signs, except in those rare cases in which there is hæmorrhage from the gums. As a rule, the tongue is clean and natural at the outset, and its subsequent state depends upon the degree of febrile excitement which may be set up, or upon the development of a typhous state, when it may become foul with various well-known aspects, or with dry and black sordes accumulating on the teeth. From the beginning of the attack the appetite for food is destroyed, whatever the state of the buccal cavity; and sometimes there is much, at others insatiable, thirst. The *bowels* are more commonly costive than the reverse. In some outbreaks costiveness has been of general occurrence. Diarrhœa, late in the disease, is not unfrequently to be attributed to the previous administration of purgatives, and involuntary stools are usually one of the accompaniments of complete nervous and vital prostration.

3. *The Urinary System.*—In the simple form of epidemic cerebro-spinal meningitis the urine does not exhibit any marked change. It may be more abundant, and slight deposits of lithic acid may occur. In the fulminant, and in severe cases of the purpuric forms, it commonly (in the first-named form perhaps invariably) contains albumen, some-

times in large amount, and occasionally cylindrical casts and blood-corpuscles. Retention or incontinence of urine has occurred in the progress of the disease.

4. *The Respiratory System.*—In all the graver cases the respiration is more or less altered. It is sighing, laboured, or interrupted. Dr. Burdon Sanderson writes of the outbreak on the Lower Vistula: "In all severe cases, whether of children or adults, the breathing was embarrassed in proportion to the general gravity of the symptoms. This embarrassment was marked by a slow laboured inspiration, followed by quick respiration and a long pause,—that condition of breathing which is so frequently observed in continued fever (especially in typhoid), and is often called suspirious. In all the fatal cases which came under my notice, the most prominent symptoms which preceded death were those which indicate impairment and perversion of the respiratory function. As the breathing became more hurried and difficult, the general depression became more intense, the pulse became weaker and quicker, and the temperature of the skin more elevated." Dr. S. Gordon records a case, fatal in less than five hours, in which the respirations rapidly fell to nine per minute, the pulse at the time being 120.

5. *The Circulatory System.*—The cardinal point with respect to the circulation, as indicated by the radial *pulse*, is defect of arterial tension. This has been common to all epidemics, with hardly an exception; and the exceptional instances have probably been more apparent than real. The frequency of the pulse does not admit of general statement. It has a wide range. In the epidemic on the Lower Vistula, the pulse in six adult cases observed by Dr. Burdon Sanderson varied from 56 to 98, the average beats being 85. In several cases noted by the same observer "its frequency varied considerably from day to day, without apparent relation to the condition of the patient in other respects." During the Philadelphia outbreak of 1866, in 98 cases observed by Dr. W. H. H. Githens, the pulse varied from the normal beat to 150 per minute in uncomplicated cases, and reached as high as 160 in two cases in puerperal women. "It was in all very weak, with a dichrotic tendency, sometimes entirely imperceptible in the radial artery, and always interrupted by slight pressure."

6. *The Cutaneous System.*—In respect of dryness or moisture or feeling to the touch, the *skin* presents no constant condition; but, in numerous cases, it is the seat of various forms of eruption of remarkable interest. The extent of prevalence or predominance of one or other of these different forms of eruption has varied considerably in the numerous recorded outbreaks. In the epidemics which have occurred in the United States, *petechiæ* have been so common as to have given rise to the popular name of the disease (*spotted fever*), and to have induced Dr. G. B. Wood, Professor of the Theory and Practice of Medicine in the University of Pennsylvania, to adopt as the technical designation of the disease the term *petechial fever*. During the recent outbreak in Ireland (1866-67), *purpura* was the

predominant form of eruption, and Professor Stokes proposed to designate the malady *malignant purpuric fever;* other observers also suggesting terms founded upon this character. In the outbreak on the Lower Vistula (1865), an herpetic eruption was most common. In all the greater outbreaks, each form of eruption mentioned in the definition of the disease has been observed; but the proportion of cases in which one or other form of eruption has prevailed has varied greatly in each outbreak. In some of the earlier outbreaks in the United States few cases occurred in which a petechial eruption was not noted. Of 98 cases admitted into the Philadelphia Hospital (Blockley) in 1866, 36 had petechiæ; 13 mixed petechiæ and erythema; 9 erythema and urticaria; 3 indistinct petechial mottling; and 37 no eruption at all (Githens). In the outbreak on the Lower Vistula the proportion of cases exhibiting an eruption was comparatively small; in the recent outbreak in Ireland, large. The forms of eruption observed are as follow:—(*a*) *Vesicles.* A vesicular eruption (*eczema,* Hirsch), sometimes herpetic in character, chiefly appearing in the vicinity of the lips, but occasionally extending over the sides of the face, diffused more or less on the trunk, or showing itself in patches on the limbs. This symptom has occasionally taken the form of shingles. It is most commonly noticed in the simple form of the disease, but it may take place in either of the other forms, and, when associated with purpura, the vesicles may be flattened and rest upon a livid base, presenting a horrible aspect. This form of eruption may appear as early as the second day. (*b*) *Purpura.* 1. True *petechiæ.* 2. *Purpuric spots,* varying in size from a split pea to half-a-crown, with more or less extensive effusions of blood, or of its colouring matter, into the cutis (*vibices, ecchymoses*). The spots have sometimes a regular, sometimes an irregular, even a ragged, outline. Their size may remain fixed from the time of their first appearance, or it may increase largely and rapidly. They may be of a light or dark red colour at the outset, subsequently becoming purple and black; or, as is most common, they may from the beginning be dark purple or black, their blackness being often fittingly likened to that of ink—the eruption resembling "spots" or "splashes" of that fluid. They may appear on the trunk or limbs only, or they may be scattered copiously over the whole surface of the body, including the face. The purpuric spots are frequently hard to the touch, the margin being defined, and giving the impression to the fingers of being raised above the surface; sometimes a vesicle forms above several of the spots, and gangrene of the adjacent tissue takes place. Dr. S. Gordon writes of the recent epidemic in Ireland: "Many cases are accompanied by a distinct eruption, which comes out with great rapidity; is found over all parts of the body, but chiefly on the lower extremities; is of a very dark colour, sometimes very deep brown, or purple, or even black. The spots are of various sizes and shapes, some small and round, others large and irregular; some appear like large spots of very black purpura, only more mottled and more irregular in colour and shape; others are more confined, and raised above the level of the skin, consisting of an effusion into its substance: many patients die in this stage, but in some the disease progresses, and these spots are absorbed, leaving a yellowish mark under the cuticle; or they pass into superficial gangrene, which was spreading at the time of the patient's death, or is healed with loss of substance." Purpuric spots are sometimes, although rarely, one of the earliest signs of the fulminant and purpuric forms of the malady; or they may occur at any period during the more advanced stages. Usually they appear at some period during the first four days, chiefly perhaps during the first or second day. Sometimes, with or without the purpuric spots, there is a cyanosed aspect of the skin, or a peculiar livid mottling. During recovery the purpuric spots gradually lose their defined character and fade away, passing through the different stages of colour which mark a healing bruise. (*c*) *Roseola, erythema, &c.* Rose-coloured spots or patches are occasionally observed; also erythema, more or less diffused, a rubeoloid eruption, and urticaria.

7. *Temperature.*—The temperature of the body, as marked in the axilla, is heightened in every case; except, perhaps, those accompanied by profound collapse from the beginning. In many cases this heightened temperature is found contemporaneously with the invasion of the disease; in other cases there is no conspicuous increment until the second or third day. When the characteristic symptoms of the malady are developed, the temperature rarely falls below 100° Fahr., and, as the disease advances, it ranges in adults from 100° to 105°, in children sometimes even higher. There is no constant or conspicuous difference between the morning and evening temperature, as in typhus and typhoid. A steady fall marks the decline of the disease and the approach of recovery; a rapid fall ushers in collapse or death.

COMPLICATIONS.—The course of the disease is liable to be modified by certain complications. Of these the chief are as follow:—(*a*) Thoracic inflammations: pleurisy, pneumonia, bronchitis, or pericarditis. Dr. S. Gordon describes œdema of the lung and diffuse pulmonary apoplexy. (*b*) Swelling or inflammation of the parotids. (*c*) Inflammation of the large joints, marked by swelling and pain, and sometimes ending in sero-purulent effusion. This complication, in its less aggravated form, has been described by some writers as rheumatic. (*d*) An inflammatory condition of the eyes and ears, as already noted. (*e*) Bed-sores. Large, deep, black sloughs occurred in four cases out of 161 treated in the Philadelphia Hospital in 1866. (*f*) The course of the disease has also been complicated by the supervention of other maladies, namely, (1) *Intermittent fever*, or certain paroxysmal phenomena simulating malarious poisoning: a complication which has led to erroneous notions of the nature of the disease. In the outbreak on the Lower Vistula cases were observed in which regular or irregular intermissions took place that could not be assigned to a malarious origin. (2) *Typhoid fever*, the two diseases prevailing simultaneously in the same district. The symptoms of both diseases more or less modified, pursue their course together, and the characteristic lesions of typhoid as well as of epidemic cerebro-spinal meningitis are discovered after death. (3) Measles and scarlet fever. (4) Cholera (Levy).

DURATION.—In the outbreak on the Lower Vistula, the most acute cases terminated fatally in from 12 to 72 hours. Cases of less intensity, but in which the patient eventually died in a typhous state, lasted from 8 to 14 days, the characteristic symptoms of the disease persisting to the end. In the more protracted, or complicated cases, from 5 to 8 weeks have passed before a patient entered upon convalescence, and death has taken place in the 6th or 7th week. Of the cases observed in the Philadelphia Hospital (1866), the duration of those which ended fatally was from 48 hours to 14 days; of those which recovered, from 20 to 30 days, the acute symptoms rarely exceeding a fortnight. In the outbreak of 1866 in Ireland, Dr. S. Gordon reported a well-marked case which ended fatally after less than *five hours'* duration. A large proportion of the fatal cases in that outbreak died in from 10 to 48 hours; in other cases the fatal ending did not occur until the close of the second and during the course of the third week of the disease. The duration of the disease, as shown by death, may be clearly stated; as marked by the beginning of convalescence, it does not admit of definite description. Moreover, convalescence is often very protracted. The course of the disease towards recovery is sometimes interrupted by *relapses.*

TERMINATION.—The disease terminates, after a longer or shorter period of convalescence, in health; or it entails during convalescence a series of physical or mental ills; or it ends in death. The rate of *mortality* of the disease is the measure of probable recovery. It varies much in different outbreaks, but is at all times formidable. Among the cases observed in the Philadelphia Hospital in 1866 the mortality was 33 per cent.; in the Hardwicke Hospital, Dublin, the same year, the mortality was 80 per cent. Dr. Stillé remarks that, "while ten epidemics in various places, occurring between 1838 and 1848, presented an average mortality of 70 per cent., a similar number occurring during the decade from 1855 to 1865 gives an average mortality of about 30 per cent. This remarkable fact would seem to indicate a gradual decline of power in the epidemic." The minimum rate of mortality recorded is 20 per cent. The proportion of fatal cases is greatest, and the duration of these cases least, at the commencement of an outbreak. The *sequelæ* which interfere with the restoration of the patient to perfect health are:—Deafness; impaired vision from structural changes in one or both eyes; paralysis of one or more limbs or of certain groups of muscles; impaired memory; carbuncles, and boils. Dr. S. Gordon describes a case in which the patient "recovered from all the acute symptoms, but gradually passed into a state of almost organic life. He ate, drank, and slept well; he passed solid fæces and urine without giving any notice, yet, evidently, not unconsciously; he was excessively emaciated, and there was a peculiar mouse-like smell from him; he seemed to understand what was said to him, but he could not answer; he never called for anything; his breathing was rather slow; his pulse, 120; his heart acting with a peculiar strong jerking motion; his eye was quite well, as also his knee [he had suffered from ulceration of the right cornea and immense effusion into the right knee-joint]; he could draw his legs and arms up to him; but he could not use his hands at all." Such was the condition of the patient fifty-eight days after the invasion of the disease.

MODE OF DEATH.—Death chiefly occurs from (*a*) asphyxia, caused by damage to the respiratory nerve-centres; (*b*) from asthenia; and (*c*) in some of the fulminant cases probably from necræmia, so profound are the changes observed in the blood.

DIAGNOSIS.—In some instances the disease approximates in certain symptoms to *typhus* or *typhoid*, and it occasionally prevails contemporaneously with both maladies. But the history of the development and progress of the disease, with the absence of characteristic eruption, will usually clear up any doubt. From *sporadic spinal meningitis* the disease is distinguished by its epidemicity, the almost constant concurrence of cerebral disorder, the tendency to cutaneous eruptions, the great mortality, and the rareness of protracted or permanent paralysis or contraction of the lower limbs. The distinction between the disease and *cerebral meningitis* is less defined as to particular symptoms, especially in children, but the mode of development of the malady will rarely leave much room for doubt during an outbreak. *Tetanus* (so-called idiopathic), with which it is suggested that epidemic cerebro-spinal meningitis may, under certain states of spasm, be confounded, never manifests the early grave cerebral symptoms which occur in the latter disease. The tetanoid contraction also observed in epidemic cerebro-spinal meningitis is rarely, if ever, as in tetanus, aggravated by sudden and painful spasms. The grouping of the symptoms in the two diseases is, moreover, altogether different. Dr. S. Gordon points out the possibility of confounding the purpuric form of epidemic cerebro-spinal meningitis with malignant measles, which malady has often prevailed at the same time. The last-named disease may resemble the fulminant form of the first-named in several respects, particularly the rapidity of development, the dark colour of the eruption, and the rapid appearance of petechiæ; also in the sudden and often extreme collapse which accompanies the invasion of the affection. But the eruption of measles rarely loses its characteristic form, and the affection of the respiratory passages is commonly present, while purpuric spots and patches are seldom observed. Dr. S. Gordon also states that he has known several cases in which the earlier symptoms of epidemic cerebro-spinal meningitis in young excitable females have been mistaken for *hysteria*. Dr. Murchison records a case which presented the symptoms of cerebro-spinal fever, including severe headache, moaning, retraction of the head, rigidity of the arms, and vomiting; but in which, after death, the duration of the case having been ten days, "no appreciable lesion of the membranes of the brain or spinal cord could be discovered, and the cause of death was ascertained to have been uræmia from contracted kidneys, and recent pericarditis."[1]

PROGNOSIS.—At the best, the prognosis of the disease is very grave. The mortality may be equally great in each of the three varieties, and petechiæ and purpura do not, as in other acute diseases, necessarily indicate an aggravated degree of danger. In 50 per cent. of the cases recorded by Dr. Githens, in one of the least fatal outbreaks known, petechiæ were present, and it is especially remarked that neither this nor any other form of eruption had "any reference to the prognosis." But when hæmorrhage into the cutis is extensive, either from the number or the size of the spots, and is accompanied by marked signs of vital prostration, it indicates an extremity of danger, although not a certainty of death. The disease is more fatal among infants and young children than among youths and adults in the prime of life; but, in some outbreaks, the latter have suffered most. After thirty years of age it becomes more dangerous. Life is most endangered in the earlier days of the disease, particularly during the first five. But danger is present at all periods of the malady, and the convalescent is not entirely safe until health is fully restored. Of the special symptoms, whether of excitement or depression, the rules of prognosis hold good which apply to other highly fatal acute maladies.

MORBID ANATOMY.—The essential anatomical characteristics of the disease, found after death, are hyperæmia, often intense, of the pia mater of the brain and spinal cord; with more or less copious sub-arachnoid and interstitial effusion into the meshes of the congested pia mater, either of serum, or of a transparent, gelatinous material, or of purulent matter: the latter more frequently than either of the two former. The purulent effusion is of greenish or yellowish colour, and is sometimes flaky. It has been found in a case in which death took place in less than five hours from the invasion of the disease (S. Gordon). The extent to which these appearances are observed and the amount of effusion varies greatly in different cases. No part of the encephalic or spinal pia mater and arachnoid may be free, or certain portions alone may be affected; but effusion is limited to the sub-arachnoid space, and does not occur into the arachnoid cavity. Under the microscope, according to Dr. Burdon Sanderson, the gelatinous material is "always found to consist of cell-like bodies, either adhering to each other so closely that they could not be completely separated, or imbedded in a transparent interstitial substance; while the sero-purulent liquid which occupied the spinal sub-arachnoid space, and in some cases the ventricles, exhibited corpuscles and granules floating freely. The cell-like bodies, although in general resembling pus corpuscles, did not present that uniformity of size and character which is met with in normal pus. They were usually, but not always, of regular circular contour, and varied in diameter from $\frac{1}{3500}$th to $\frac{1}{1200}$th of an inch. Occasionally they exhibited the appearance of an external cell-membrane, but in most instances this could not be made out even in perfectly fresh exudations—as, *e.g.* in those cases which were examined as early as eight hours after death. They invariably contained numerous granules, some of which were cleared away on the addition of acetic acid. Those which remained were highly refractive, but did not assume any special form of arrangement. The interstitial substance was beset with granules, some of which were albuminous, others fatty. It was most abundant and distinct on the surface of the spinal arachnoid, where it infiltrated the fine connecting tissue and minute blood-vessels of the pia mater."

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[1] Proceedings of the Pathological Society, vol. xviii.

For the rest, the nervous system of the brain and spinal cord is usually gorged with blood, unless death has taken place late in the course of the disease. The visceral arachnoid is frequently thickened and opaque. Dr. Klebs[1] has shown that often, where the eye detects opacity alone, the microscope reveals extensive cell-formation, purulent in character. Softening of some portion of the spinal cord has sometimes been observed; and Mr. J. Simon thinks that, "for practical purposes, the state of the covering membranes of the nervous centres may be regarded as a mere index of changes more or less distinctive, which those centres in their own intimate composition have at the same time undergone; and hence it is that the essential phenomena of the disease during life consist in disturbances, more or less grave, of the functions of these all-important organs." Dr. Klebs describes œdema of the medullary substance and loosening of the nervous elements, and suggests that this may explain the extensive motor disturbances which have been observed in some cases, in which purulent effusion into the subarachnoid space has been slight. The same author has observed also purulent encephalitis.

In fatal cases of the simple and purpuric forms of epidemic cerebro-spinal meningitis the characteristic anatomical lesions are almost invariably found. In the fulminant form of the disease they are often absent. The cases in which there is no indication of morbid change in the nervous centres are exceedingly few. It has been suggested that in these cases death has occurred so rapidly that there was insufficient time for the formation of a structural lesion. In connexion with this explanation the case recorded by Dr. S. Gordon must be borne in mind, in which purulent effusion was found, although the whole duration of the attack was under *five* hours.

[1] Zur Pathologie der Epidemischen Meningitis. Virchow's Archiv, Band xxxiv. British and Foreign Medico-Chirurgical Review, 1868.

Practically the apparent absence of characteristic anatomical change in the nervous centres, in certain rare cases of epidemic cerebro-spinal meningitis, is a phenomenon analogous to that which sometimes occurs in rapidly fatal cases of malarious, variolous, and scarlatinous poisoning, in which the characteristic eruptions or lesions of the diseases have not been developed.

No lesions manifestly peculiar to epidemic cerebro-spinal meningitis have, as yet, been found in other organs of the body. Such lesions as occur elsewhere than in the coverings of the brain and spinal cord usually have a definite relation to the thoracic, abdominal, or genito-urinary complications which may have happened during the progress of the malady. Dr. Klebs, however, describes certain changes in the intimate structure of the kidneys and liver, which he believes to be characteristic of the disease. In the fatal cases of the purpuric form of the affection recorded by Dr. S. Gordon, and other writers, an excessive fluidity of the blood was noted.

History and Geographical Distribution.—The scientific history of epidemic cerebro-spinal meningitis dates only from the fourth decennium of the present century. At that period the disease was, for the first time, clearly distinguished as an independent malady; and with the light then obtained, outbreaks which had occurred earlier in the century, in various localities of both the Eastern and Western hemispheres, and had been recorded under other names, were recognised as of a similar character. It has been sought, indeed, to show that epidemic cerebro-spinal meningitis has probably existed from remote periods (Tourdes, Boudin). The probability may be admitted, for the first recognition of a malady as an independent affection does not necessarily imply that the malady is new.

In 1837 epidemic cerebro-spinal meningitis broke out in the south-west of France, and prevailed in various localities of the district intervening between Bayonne and La Rochelle, and along the whole line of the Pyrenean frontier. Dax, Bordeaux, Auch, Foix, Narbonne, and Perpignan suffered, as well as the two cities previously named. The disease, according to Boudin, at the commencement and during the continuance of this outbreak, chiefly showed itself among troops in garrison. During 1837 and 1838 the garrisons of Bayonne, Dax, Bordeaux, Rochefort, and La Rochelle suffered. From 1838 to 1841 the disease was prevalent among the garrisons of south-eastern France, particularly those of the valley of the Rhone. Thus it broke out at Toulon, Marseilles, Aigues-Mortes, Nîsmes, Avignon, and Pont-Saint-Esprit. In the course of the four years 1839-40-41-42, the malady appeared in succession among the troops occupying the fortresses of Strasburg, Schelestadt, Colmar, Nancy, Metz, and Givet. From 1839 to 1842 it prevailed among the forces at Versailles, Saint-Cloud, Rambouillet, and Chartres. Those stationed along the coast of Brittany, at Brest, L'Orient, Nantes, and Ancenis, suffered in 1841; and during 1840 and 1841 the disease manifested itself among divers detachments of a regiment scattered at Laval, Le Mans, Château-Gontier, Tours, and Poitiers. It was during the outbreak of which the most remarkable episode is thus sketched by Boudin that a scientific knowledge of epidemic cerebro-spinal meningitis was first obtained. From 1837 to 1848 inclusive, forty-seven outbreaks of the malady were recorded in thirty-six of the eighty-six departments into which France was then divided. These outbreaks were distributed in the departments of the Loire, Rhône, Bouches-du-Rhône, Bas-Rhin, Seine, Seine-et-Oise, Landes, Basses-Pyrénées, Charente-Inférieure, Gard, Vaucluse, Var, Moselle, and Loiret. The three first-named departments suffered most. In 1840 the disease appeared in Naples and prevailed in the Papal States. The same year it broke out among the French garrison at Douera, Algeria, and during the next seven years it attacked numerous towns and localities of the province, affecting the civil population, both European and native, as well as the military. In 1844 an outbreak of the disease took place among the civil population of Gibraltar; and in 1846 the malady showed itself slightly in Ireland among the inmates of the Rathdown, South Dublin, and Belfast workhouses, and several cases occurred among the population of Dublin. During 1849 and 1850 the disease was prevalent to some extent among the French troops in Italy, and in the last-named year several localities of France suffered from it. Epidemic cerebro-spinal meningitis appeared in Denmark in 1841, and prevailed in that country until 1848. The disease was first noticed in Sweden in 1854, this country again suffering from it in 1861. In Norway the malady broke out in 1859, and it prevailed in that country more or less until 1867, if not to a later period. During 1860 the disease was prevalent in Holland; and the same year it was widely spread in Portugal. In 1863, 1864, and 1865 an extensive outbreak occurred in North Germany; and in 1866 the malady broke out in Dublin and elsewhere in Ireland. Cases were recorded in St. Petersburg during 1866 and 1867.

In the United States (where the disease may be traced back to the commencement of the century), epidemic cerebro-spinal meningitis became prevalent about the same time that it exhibited great activity in Europe. From 1842 to 1850 inclusive, a series of outbreaks took place in the States of Kentucky, Tennessee, South Illinois, Mississippi, Arkansas, Alabama, Pennsylvania, Massachusetts, New York, and North Carolina. After this period there would appear to have been an interval of comparative inactivity. In 1861 the disease broke out in North and Central Missouri, and from that time to the present it has prevailed, more or less extensively, in almost all, if not all, the States of the Union, with the exception, perhaps, of the Pacific States. In 1862 outbreaks were recorded in Connecticut, Kentucky, Indiana, and Tennessee; in 1863, in Rhode Island; in 1864, in Pennsylvania, Ohio, Illinois, New York, Maryland, Massachusetts, and Vermont; and in 1865, in North Carolina and other Southern States. During 1867 and 1868 the disease was active in several States.

It must be borne in mind that these historical notes very imperfectly represent the probable prevalence and geographical distribution of the disease. They simply include a brief summary of outbreaks which have come under the notice of thoughtful observers who have published their observations. The history of the malady in the British Islands is, perhaps, less liable to error from this source. The earliest recorded outbreak of the disease occurred in Ireland during the early months of 1846. It broke out to a very limited extent among the boys living in the Rathdown Union, South Dublin, and Belfast workhouses; and two cases, both in females, one aged 17 years, the other 36 years, were admitted into the Hardwicke Hospital, Dublin.[1] Prior to this outbreak, there is not any trustworthy history of the presence of epidemic cerebro-spinal meningitis in the British Islands. It is not improbable, however, that the disease existed at Blackaton, in Devonshire, in 1807;[2] and at Sunderland in 1830.[3] Dr. Benajamin W. Richardson saw, he believes, a case at Mortlake, Surrey, in 1843.[4] From the time of the outbreak in 1846, cases of a similar malady were occasionally observed in Dublin, until the latter half of 1850, when they became more common.[5] There is no further notice of epidemic cerebro-spinal meningitis in Ireland until the year 1865, when cases began to be again observed in Dublin.[6] A case of cerebro-spinal meningitis was observed by Dr. Samuel Wilks, in each of the three years 1856, 1858, 1859, in the metropolis.[7] In October 1859, a fatal case of cerebro-spinal disorder, with petechial eruption, came under the notice of Dr. Henry Day, in the vicinity of Stafford. In this case, hyperæmia of the meninges of the brain and spinal cord, and copious effusion of fluid at the base of the brain, were discovered after death. A similar but more rapidly fatal case was also observed by Dr. Day, in the Stafford General Infirmary in September 1865.[8] The largest and most fatal outbreak of epidemic cerebro-spinal meningitis which has occurred within the limits of the United Kingdom began in Ireland in March 1866, and attained its chief development in the subsequent winter. Its effects were almost entirely limited to the sister island, and the brunt of the outbreak fell upon Dublin. Other localities affected, during the first year of prevalence, were Tullamore, Parsonstown, Mitchelstown, Thurles, Clondalkin, and the Curragh camp. Subsequently cases were recorded in the counties of Cork, Waterford, Clare, Galway, Meath, Down, &c. The cases were not very numerous in Dublin; and in the country towns they were comparatively few. It is noteworthy that, as in the earlier outbreaks in France, the military in Ireland, in proportion to their strength, suffered prominently from the disease. In some of the country districts cases were recorded among the troops alone, or among persons in immediate connexion with them.[1] In January and February 1867, an outbreak of a disease characterised by severe rigors, tetanic convulsions, intense neuralgic pain in the head and upper part of the trunk, increased sensitiveness of the surface, obstinate vomiting, restlessness, and, in one instance at least, by a dark purple eruption, but of which not a single case died, took place at Bardney, in Lincolnshire, a village about ten miles east of Lincoln, on the verge of a fen country, and having a population of 1,500, the bulk of whom are engaged in agricultural pursuits.[2] Two cases of epidemic cerebro-spinal meningitis were recorded in London in the summer of 1867. One, a case of the fulminant form of the malady, in which death occurred in twenty-seven hours, took place in June;[3] the other, a case of the purpuric form, in which death occurred in seven days, took place in July.[4] Since the attention of English practitioners has been more fully directed to epidemic cerebro-spinal meningitis by the late outbreak in Ireland, so-called sporadic instances of the disease have been noted in various parts of the kingdom, under circumstances which lead to the surmise that the malady is not so rare among the population as had previously been supposed.

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[1] Dr. Robt. Mayne, Dublin Quarterly Journal of Medical Science, 1846, vol. ii. p. 95.

[2] Mr. Henry Gervis, Medico-Chirurgical Society's Transactions, vol. ii.

[3] Dr. John Scott, Medical Times and Gazette, 1865, vol. i. p. 515.

[4] Social Science Review, May 1865, p. 398.

[5] Dr. McDowell, The London Journal of Medicine, 1851, vol. iii. p. 858.

[6] Dr. Kennedy, The Medical Press and Circular, June 12, 1861, p. 551.

[7] The Lancet, April 15, 1865, p. 389.

Clinical Histories and Comments, 1866, pp. 3-7.

The peculiarity of distribution of the disease in the British Islands, its epidemic manifestations being limited to one portion of the kingdom, and chiefly, even in recurrent outbreaks, to a small section of the population of that portion, is not an isolated phenomenon. Notwithstanding the wide geographical prevalence of the malady as shown by the foregoing details, it must not be concluded that this prevalence represents a general diffusion of the disease among the different populations during the periods of its activity. The outbreaks of epidemic cerebro-spinal meningitis, as a rule, are limited to small sections of a population, and its distribution is by a series of isolated outbreaks, rather than by extensive spreading. This was shown remarkably, as already described, during the outbreak in France in 1837 and following years, when the ravages of the malady were principally confined to certain garrisons, and even to small sections of a garrison, without affecting the surrounding population. A like limitation of the disease to certain detachments of troops was observed during the recent war in the United States; and the restriction of the malady to small portions of workhouse populations, as in the first outbreak in Ireland, is an analogous phenomenon. Perhaps the sole outbreak in which an extensive diffusion of the disease among a community has occurred was that in the province of Dantzic, in 1864-65. The tendency to reproduction in a locality, as in Dublin, was particularly observed during the great outbreak in France from 1838 to 1848, when the disease reappeared again and again among the forces in Bayonne, Versailles, and Avignon, notwithstanding changes of garrison. The freedom of England and Scotland from epidemic outbreaks of so widely spread a malady is very remarkable; particularly if the seeming occasional cases of the disease to which reference has been made are to be regarded as true examples.

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[1] Dr. E. D. Mapother, and Staff-surgeon Dr. Jeffrey A. Marston, The Lancet, July 6, and July 13, 1867; also Transactions of the Epidemiological Society, vol. iii. p. 118 and p. 129.

[2] G. M. Lowe, M.B. The Lancet, June 26, 1867, p. 790; Mr. Geo. Newnham Woolley The Lancet, Aug. 3, 1867, p. 130.

[3] Dr. Edward Crisp, The Lancet, June 22, 1867, p. 773.

[4] Dr. Thomas Clark, The Lancet, July 13, 1867.

ETIOLOGY.—(*a*) *Predisposing Causes.—Age.* The personal liability to the disease is not governed in any definite manner by age. In some epidemics children, in others young people, in others again adults of from thirty to fifty years, have suffered in greatest proportion.—*Sex.* Generally, and in some outbreaks very markedly, *males* are more liable to the disease than *females.—Profession.* During the outbreaks of the disease in France from 1837 to 1849, a peculiar proclivity to the disease was observed among soldiers. But in subsequent outbreaks in France, and wider-spread outbreaks elsewhere, no special liability to the disease was manifested among any vocation.—*Climate and Seasons.* In the Eastern hemisphere our knowledge of the disease is limited to Western and Central Europe and Algeria, the northern boundary of the district not passing beyond lat. 61° N., the southern not beyond lat. 35° N.—the one extreme closely approaching the arctic, the other the torrid zone. In the Western hemisphere the records of the malady are confined to the populous districts of the eastern division of the United States, from lat. 30° N. to lat. 48° N. It is noteworthy that the northern and southern limits of distribution in both hemispheres but slightly overlap the isothermal lines 5° and 20°. *Season* acts as an unquestionable and powerful predisposing cause of epidemic cerebro-spinal meningitis, which is especially a disease of the cold months. Of 216 local outbreaks in France and the United States, 166 prevailed between December 1st and May 31st; 50 in the other six months of the year. In Sweden, of 417 local outbreaks, 311 took place in the former period of the year, 106 in the latter (Stillé). During the recent outbreak in Ireland, the brunt of the disease fell between January and July 1867. Of 85 outbreaks in various parts of Europe and the United States, noted by Hirsch, 33 prevailed in winter, 24 in winter and spring, 11 in spring, 1 in spring and summer, 2 in summer, 1 in summer and autumn, 1 in autumn, 1 in autumn and winter, 3 in autumn, winter, and spring, and 6 prevailed throughout the whole year.—*Locality and soil* do not, so far as yet ascertained, exercise any manifest influence over the disease. It has been observed indifferently on low grounds, on highlands, and on soils of the most various character.—*Sanitary conditions.* No definite relation exists between the occurrence of the disease and the sanitary state of habitations or of individuals. It has prevailed in some epidemics alike among the affluent and the impoverished—among those who are well-fed, wellhoused, and well-clothed, as among those who are ill-fed, ill-housed, and insufficiently clothed. In certain outbreaks, as in that on the Lower Vistula, the prosperous classes suffered to a much less extent from the malady than the poor and miserable, who were subjected to privation and to much foulness of persons, dwellings, and atmosphere.

(b) *Exciting Causes.*—Fatigue has been mentioned as an exciting cause. In some of the early outbreaks of the disease among French troops, France being at war at the time, fatigue apparently exercised a determining influence. Again, during the recent outbreak in Ireland, the malady appeared very early among a "flying column" of troops occupied in the suppression of the Fenian disturbance, and exposed to great fatigue and inclemency of weather. But fatigue has played little or no part in determining the disease among the civil population, especially among children and the inmates of workhouses and prisons.—*Cold.* The marked predominance of the disease in the winter and spring months has suggested a causal connexion with cold. Hirsch has submitted the question to a detailed examination, and with this result: that, although we cannot exclude the suspicion that the temperature of winter and spring may have some direct effect upon the genesis of the disease, "the modifications in the mode of living incidental to these seasons exert, in a far higher degree, an influence favourable to the presence of this as of many other infectious maladies."[1] —*Certain Insanitary States.* There is not any constant or even common relationship between any insanitary state and the appearance of the disease. Neither foulness of house and its surroundings, nor of the atmosphere, whether from putrid emanations or from overcrowding, nor impurity of any other kind, has any determinate relation with epidemic cerebro-spinal meningitis. But Hirsch remarks[2] of the outbreak in the province of Dantzic in 1865, that "the disease prevailed exactly in that season of the year in which, on account of inclement weather, many individuals were crowded together into small and dirty rooms kept constantly closed by their occupants, and from which all ventilation was excluded, and in which the before-mentioned unfavourable hygienic conditions [dampness, great filth, and an atmosphere loaded with putrid emanations] were extremely perceptible." The causes here suggested have been held to be not altogether inoperative in other and more circumscribed outbreaks.—*Communication of the sick with the well.* The great majority of observers have come to the conclusion that the disease is incommunicable from the sick to the well. Among the minority who hesitate to accept this deduction without reservation are Professor Hirsch, Professor Stokes, and Mr. J. Simon. The facts which suggest the possibility of the active cause of the disease being portable in some way are of the following character:—(*a*) A child was seized with epidemic cerebro-spinal meningitis, and died. A second child of the same family was attacked with the malady a few days later. The day following the attack of this child, the mother, who slept in the same bed with it, sickened of the disease.[1] (*b*) 1. On the 8th February, 1865, a youth, aged 20 years, was attacked with the characteristic symptoms of epidemic cerebro-spinal meningitis. He was nursed by a woman from another village. The youth died, and after his death the woman returned home. She soon sickened, and she died of the epidemic disease on the 26th February. There had been but one case previously in the village. To the interment of the woman came a family from another locality, the funeral obsequies, as customary in the district, being performed with the coffin open. After the return home of this family, a child, three months old, sickened immediately of meningitis and died within twenty-four hours. Then a man who had accompanied the family to the interment was attacked with the disease, and died on the 2nd of March. Lastly, a girl, in the same locality, who had also been at the funeral, was seized, and died on the 7th March. 2. At another village, two children of one family, aged three and a half and one and a half years respectively, died of the epidemic, one on the 27th January, the other on the 7th February. The clothes of the deceased were taken to a neighbouring village, and came into the possession of a girl aged five years. She soon sickened of the epidemic, and died on the 14th February.[2] (*c*) Boudin relates instances of the appearance of the disease in garrisons, and among the civil population of towns, after the introduction of detachments of troops among whom the disease had prevailed or was prevailing at the time.

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[1] Transactions of the Epidemiological Society, vol. ii. p. 369.
[2] Ibid. vol. ii. p. 372.

The foregoing facts simply suggest the possibility of the active cause of epidemic cerebro-spinal meningitis being communicable by the sick to the well. This possibility, notwithstanding the apparent formidable array of facts to the contrary, is not to be lightly dealt with. The lesson taught by the difficulties and doubts which beset the discovery of the communicability of typhoid fever and of cholera, will have been strangely misunderstood if it is necessary to urge upon observers, the importance of keeping the question of the possible communicability of epidemic cerebro-spinal meningitis constantly before the mind. In the consideration of this question, however, a caution is needed. The term " contagion " is used too indiscriminately. It has been so long employed to express the manner of transmission of disease which is witnessed in small-pox, scarlet fever, or typhus, that it is difficult to dissociate the idea of this manner from the word. It is almost impossible, in reading the opinions of those writers who have come to the conclusion that epidemic cerebro-spinal meningitis is not a "contagious" disease, to avoid the suspicion, from their use of the adjective, that they have looked upon the question too exclusively from the point of view suggested by the diseases named. It is obvious that contagiousness of a like character to that of small-pox, scarlet fever, or typhus, is not possessed by the malady under consideration. The question is: Does epidemic meningitis, like typhoid fever or cholera, possess a peculiar contagiousness of its own, a property of communicability peculiar to itself? This has yet to be solved. Another explanation of the facts which appear to indicate a possible communicability of the disease from the sick to the well is, however, open, and is set forth in the next paragraph.

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[1] Professor Stokes, The Medical Press and Circular, June 19, 1867, p. 581.

[2] Hirsch, Transactions of Epidemiological Society, vol. ii. p. 373.

Diseased grain.—Dr. B. W. Richardson has suggested that epidemic cerebro-spinal meningitis may possibly arise from the consumption of diseased grain after the manner of ergotism, and perhaps acrodynia. He thinks that the probabilities are altogether in favour of the suggestion, that "the cause in fact is a diseased grain, or fungus, contained in some kinds of flour out of which the bread-stuffs are made. This fungus may not be present in large quantities, and many persons may eat of the food without getting a poisonous part; but one will get it out of a number, and this without any communication beyond the breaking of bread together: the disease may occur in one member of a family, leaving the rest free, and in this irregular way it may be distributed, in an epidemic form, over a large surface of country." He adds, "If my hypothesis, as regards cause, be correct, there is little danger of the disorder extending widely in this country; for of our cereals used as food, nearly the whole of the population now select wheat, and our wheat generally is selected for the market with great judgment and circumspection. Any cases, therefore, that might occur would be isolated, and would be easily traced out and prevented."[1] This suggestion opens out an altogether new field of inquiry respecting the origin of the disease, and it demands active and thoughtful consideration in subsequent outbreaks. Dr. H. Day, of Stafford, has endeavoured, by experiments on the lower animals, to obtain some light on the subject. He fed three rabbits with unsound grain (wheat, oats, ergot of rye, and mouldy bread) with this result: In all the animals a spasmodic affection was produced, and in two inflammatory changes in the right eye, proceeding in one case to ulceration of the cornea, and evacuation of the contents of the globe. One of the rabbits died on the eighth day, the other two were killed on the twelfth day, and in all more or less congestion of the membranes of the spinal cord was found on dissection.[2]

The sum of our knowledge of the etiology of epidemic cerebro-spinal meningitis is this; that the clue to its explanation has not as yet been discovered.

NATURE.—1. *Is the disease malarious, as suggested by some writers?* The outbreaks in which the disease has occurred in malarious districts, or in which the malady has shown an intermittent character, are too few in number to admit of much, if any, doubt resting upon the answer. There is no sufficient ground for believing that the malady is of malarious origin. The numerous examples of prevalence of the disease in localities free from malaria set the question aside definitely. Even when intermissions or remissions have been observed in the progress of the malady, it must not be hastily assumed that they are consequent upon malarious poisoning. Hirsch has shown that certain cases of epidemic cerebro-spinal meningitis, distinguished by intermissions and remissions, which came under his own observation, took place in the course of an outbreak in a district free from malaria. Further, he states that this outbreak prevailed at a season (winter) and in a state of climate (intense cold) which notoriously exclude the prevalence of malarious diseases, even where endemic; that the period of life (1—5 years) least liable to malarious disease furnished the largest contingent of victims, while the classes most advanced in life, and who are most liable, escaped the epidemic in a remarkable degree. Finally, the infallible test of malarious disease, quinine, by its inutility in cases of the epidemic which assumed an intermittent or remittent character, showed the non-malarious nature of the affection.[1] 2. *Is epidemic cerebro-spinal meningitis a form of, or allied to, typhus?* Epidemic cerebro-spinal meningitis differs from typhus in the aspect of the patient, progress of the disease, range and course of temperature, form of cerebral affection, character of eruption, sequelæ, rate of mortality, anatomical lesions, and manner of dissemination. Differing in all essential particulars, doubt can only arise when the two diseases prevail together. Under such circumstances, cases of the fulminant and purpuric forms of the one malady may be difficult to discriminate from the graver and more rapidly fatal forms of the other. Doubt also may arise when in the course of the former disease typhous or typhoid symptoms occur. But such a doubt applies equally to the discrimination of the disease from measles and typhoid fever, as from typhus. 3. *Is epidemic cerebro-spinal meningitis a true or a pseudo-epidemic disease?* Is this disease a true epidemic disease in the sense of its being due to a specific, febrile poison (to which class of diseases the term epidemic is now well-nigh alone restricted)? Or is it a pseudo-epidemic malady, as being an exaggerated and more prevalent form, from certain climatic or other conditions, of an idiopathic inflammatory affection of the brain and spinal cord? No absolute distinction can be drawn between sporadic cerebro-spinal meningitis and the epidemic malady of the same name. But there are certain broad and well-defined differences. The conjoined inflammatory affection of the covering membranes of the brain and spinal cord, which is the rule in epidemic cerebro-spinal meningitis, is a rare exception in sporadic inflammation of the envelopes of the central nervous centres. Again, the indications of blood-change which are so common in the epidemic disease have been witnessed only in exceptional cases in the sporadic disease. The question arises whether the exceptional cases are instances of idiopathic cerebro-spinal meningitis or of the epidemic form of the malady. This cannot be determined arbitrarily. It is certain that cases of cerebro-spinal meningitis, indistinguishable from the epidemic disease, are observed, in the intervals of prevalence of the latter, even in this country (H. Day, Wilks, &c.). These cases are of much, although as yet of indeterminate interest, in reference to the etiology of the malady. It has been suggested that the blood-change and herpetic and purpuric eruptions may be of nervous origin, and consequent upon the profound alteration in the nervous system. It has been suggested, also, that the purpuric eruption of epidemic cerebro-spinal meningitis may be one of several signs of a general tendency to purpura in disease at the time of prevalence, and merely an incidental phenomenon of the epidemic malady. Thus, in Dublin (1866-67), purpura had been observed in rheumatic fever,[1] and there was an outbreak of purpura among swine,[2] contemporaneously with the epidemic. The first suggestion touches a very curious question, which as yet does not admit of solution. But it is worthy of remark that the form of eruption which of all others is peculiar to epidemic cerebro-spinal meningitis is the *herpetic,*—a form which, in some of its manifestations at least, as in herpes labialis, and in shingles, has singular neurotic relations. Mr. Jonathan Hutchinson has propounded the riddle, *Is herpes zoster an exanthem or neurosis?* [3] This is certain, that it is a symptom which has some definite connexion with lesion of nerve trunks, if not of nerve *centres.*[4] The facts upon which the second suggestion is based are of interest, but they form too narrow a basis for conclusions. 4. *Is epidemic cerebro-spinal meningitis a disease, sui generis?* The association of symptoms shows that it is an independent malady; the aptitude to blood-changes in the course of the disease, judged by analogy with like changes which occur in acute specific diseases, suggests the inference that it also is dependent upon a specific poison, from whatever source derived. This is the conclusion which appears to have the highest degree of probability in the present state of our knowledge.

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[1] Social Science Review, May 1865, p. 403.

[2] Clinical Histories and Comments, pp. 18—23.

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[1] Transactions of the Epidemiological Society, vol. ii. p. 377.

An intercurrent question arises here: Is the fulminant form of epidemic cerebro-spinal meningitis really a variety of the disease, or a different malady altogether? Dr. R. D. Lyons maintains that during the prevalence of the epidemic in Dublin in 1866, two independent diseases existed. The one, characterised by collapse, profuse purpuric eruption, great rapidity of course, excessive fatality, and absence of anatomical lesion in the nervous centres after death, he designates *febris nigra;* the other was the disease commonly known as cerebro-spinal meningitis. But it is to be remarked that the two varieties of disease have never been observed except in the same epidemic; that they pass by insensible grades the one into the other; that the most highly developed symptoms of the so-called *febris nigra* sometimes occur together with the most marked symptoms of cerebro-spinal meningitis; and that it is more consistent with experience to consider the two series of symptoms as indications of one and the same malady, rather than two maladies going forward at the same time in the same patient. A second intercurrent question is, whether the purpuric form of the disease be of scorbutic origin? The question amounts to little more than a suggestion. There are no facts which support an affirmative answer; for, apart from other well-known signs, purpuric spots are not indications of a scorbutic taint.

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[1] Dr. Banks, The Medical Press and Circular, June 19, 1867, p. 580.

[2] Dr. Mapother, The Lancet, July 13, 1867, p. 39.

[3] London Hosp. Reps. vol. iii. p. 70.

[4] Dr. H. Day (Lancet, vol. i. 1867, p. 731) expresses the opinion that an eruption would probably be more commonly found in cases of sporadic cerebro-spinal meningitis if it were more carefully looked for; and he remarks that the petechial spots which he has observed in cases coming under his own notice, sometimes do not appear until after death. My own observations coincide with those of Dr. Day. In connexion with this subject it is well to bear in mind Trousseau's so-called *cerebral* or *meningeal macula* (Lectures on Clinical Medicine, Bazire's Trans. vol. i. p. 459), a phenomenon of wider occurrence in diseases of the central nervous system than even the distinguished Professor suspected. I may add, that a short time ago I was present at the examination of the body of a patient who had died from syphilitic disease of the right hemisphere of the brain. I was particularly struck with a peculiarity in the after-death lividity which seemed to me not uninstructive with regard to the purpuric forms of cerebro-spinal fever. Life had been extinct eighteen hours. The greater part of the depending portions of the body was ecchymotic; but, in addition, many livid, circular spots, of about the size of a split pea, a few of larger size, were scattered over the upper aspect (dorsal) of the feet, and of the legs beneath the knees; also, over the upper aspect (dorsal) of the hands, and of the arms beneath the elbow. These spots, had they been observed in a case of cerebro-spinal meningitis, would have been designated "purpuric."

TREATMENT.—*Prophylactic.*—Ignorance of the true etiology of the disease limits our preventive efforts to general sanitary measures, applicable to all epidemic diseases, for the purification of houses and localities. Mr. J. Simon, recording the conditions under which the disease has prevailed, writes: "I am strongly of opinion that the best sanitary precaution which in the present state of knowledge can be taken against the disease, must consist in care for the ventilation of dwellings." He adds, however, "that in some cases, according to local reports, the distribution of an epidemic has very decidedly not been governed by conditions of overcrowding and ill-ventilation." Dr. B. W. Richardson's suggestion as to the cause of the disease should lead to the careful microscopic examination of all bread-stuffs and farinaceous preparations in use among families and communities where the disease breaks out, and the disuse of such as may be of doubtful character.

Curative.—The treatment of epidemic cerebro-spinal meningitis is as unsatisfactory as that of cholera. The evidence of the course of the disease having been beneficially affected in any outbreak by the administration of medicine is very doubtful. The too common rapid progress of the malady to death, as in cholera, and the nature of the lesions determining death, necessarily set at nought efforts to check it; medicine not being guilty either of inaptitude or inactivity. The control of this disease, as of cholera or trichiniasis, is a question of preventive rather than curative treatment, and must depend upon the discovery and limitation of its cause. In the earlier outbreaks, epidemic cerebro-spinal meningitis was treated, as an acute inflammatory affection, by bleeding and purgatives, with the general result that the fatality of the malady was probably almost invariably augmented. During the outbreak of 1866 in Philadelphia, it was found that, in the more sthenic cases, cupping the nape of the neck was "of essential service in mitigating, and generally, indeed, in wholly removing the neuralgic pains which form so prominent and so severe a symptom in many cases of the disease" (Stillé). When the state of the patient forbade the abstraction of blood, dry-cupping used in the same locality afforded signal relief, and rendered the effects of vesication more prompt and complete. This was the experience in one of the least fatal outbreaks recorded. The experience of the majority of epidemics has been against any blood-letting, local or general. The deduction to be derived as to depletion from the general state of the circulation, entirely coincides with the results of practice. For, as a rule, the pulse from the very outset contra-indicates the withdrawal of blood; and, if in any case it should seem from the general symptoms that depletion might exercise some control over the central mischief, a thoughtful regard should be given to the future. The application of *cold* to the head and spine, either by means of ice or a freezing mixture, in Esmarch's india-rubber bags, is not open to the same objection as blood-letting, and has furnished by far the most satisfactory results of all direct treatment of the acute cerebro-spinal symptoms. In its use care should be taken not to prolong the application so as to depress, or increase the depression already existing of, the whole system. When the acute nervous symptoms are accompanied by marked prostration, it is advisable during the application of the ice to swathe the limbs in hot flannels, to pack the legs and thighs with hot-water bottles, or bags filled with hot sand or salt, and to cover the abdomen with thick layers of flannel or cotton-wool. From the very outset of the disease, care should be taken to economise the temperature of the body, and anticipate its fall; and in cases characterised by collapse, or much vital depression, the application of external heat in the manner just suggested is a cardinal point of treatment. Of medicaments directly addressed to the nervous symptoms, *opium* is the most valuable. It is especially indicated when there is much restlessness, acute delirium, sleeplessness, hyperæsthesia, or painful spasm. *Morphia* is the best form of administration, and subcutaneous injection perhaps the best mode. The drug should be given in decided and frequently repeated doses, and carefully watched. Stillé says of its use during the late outbreak in Philadelphia: "We were in the habit of giving one grain of opium every hour, in very severe, and every two hours in moderately severe cases, and in no instance was produced either narcotism, or even an approach to that condition. Under the influence of the medicine the pain and spasm subsided, the skin grew warmer, and the pulse fuller, and the entire condition of the patient more hopeful. It seemed probable, however, that the full benefit of the opium treatment could be received by those only who were subjected to it in the early stages of the attack. Direct experience is here in perfect accord with the expectation which a knowledge of the pathological processes involved in the disease would naturally suggest."

A Committee of the American Medical Association has reported favourably of the *sulphate of quinia* in large doses, given at the very beginning of the disease. In some instances the drug seemed to abort the attack. The committee speaks also of favourable results reported from the combined use of *ergot* and *chloride of iron.* Some American physicians have given *ergot* in combination with *belladonna* and *belladonna* in combination with *quinine,* but with equivocal benefit. *Mercurials* have been freely used, particularly in the form of *calomel,* but their effect has been most questionable, except as purgatives. Their indiscriminate use is to be utterly condemned, and their use at all to be discountenanced. A host of other medicaments have been made use of, of which it is requisite to note only *iodide of potassium, bromide of potassium,* and *arsenite of potash.* The circumstances under which the two former drugs have been used, and are most likely to prove beneficial, will suggest themselves to the practitioner. It does not appear that any decided good has arisen from their administration. In protracted cases of convalescence the arsenite of potash may prove a valuable remedy.

Of the general treatment of the patient the hot bath (102°—106°) is, when practicable, the most important feature. The Committee of the American Medical Association recommend, when the surface is cold, friction with hot, coarse towels, or even with warm oil of turpentine, after the bath. The *regimen* should be generous and nutritious from the beginning of the disease. In the acute stages soup of some kind or other, or milk, is needed; and as soon as appetite returns, *solid* viands of any digestible character must be given. In the graver cases, when there is much restlessness and spasm or stupor, and food cannot be given by the mouth from the patient's refusal or inability to swallow, an attempt should be made to administer it by the rectum: when there is much thirst, the patient's fierce desire for drinks may be freely indulged. The state of the pulse is the principal guide to the use of *stimulants.* Their administration as a special remedy independently of the indications which generally govern their use has not been followed by good results; but they are called for when the condition of the pulse and the aspect of the patient show manifest flagging of vital power. The *sequelæ* of the disease must be treated on ordinary principles.

Too frequently the state of the patient as to delirium, spasm, and irritability of the stomach limits the use of medicine to subcutaneous injections, prevents the proper administration of food, and restricts even the application of external measures. To this unhappy combination of unfortunate and uncontrollable conditions the inefficiency of treatment may partly be attributed.

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THE PLAGUE.

By Gavin Milroy, M.D.

Definition.—The Plague may be briefly defined to be a fever, usually of an adynamic type, accompanied with bubos, carbuncles, and petechiæ.

Synonyms.—This is the λοιμός of Hippocrates and Galen; the Pestilentia of Celsus and other Roman writers; the Pestis; Typhus pestilentialis; Typhus gravissimus; Typhus bubonicus, &c. of many nosologists. It is the Febris Adeno-nervosa of Pinel, and the Peste Orientale, Typhus d'Orient, of other French authors; the Black Death, Levant Plague, Pestilential Fever, of English writers. Dr. Copland terms it the Septic or Glandular Pestilence.

Symptoms.—The bubos may be in the groins, axillæ, or the neck; occasionally, but very rarely, the popliteal glands have been affected. The carbuncles are generally on the upper or lower extremities—most frequently on the legs, but sometimes on the chest, back, or cheek. Their number may be from one or two to a dozen or more, and they vary much in size and in the tendency to become gangrenous. The petechiæ and vibices may be scattered over every part of the body. The pyrexial symptoms of the Plague differ in no respect from those in other forms of pernicious or malignant fever. There are the usual prodromal phenomena of lassitude, rigors, nausea, headache, and vertigo; oppression about the præcordia, anxiety and restlessness, with a heavy stupid expression of countenance, and a muddy or suffused state of the eyes. Then follow heat of the skin and great thirst, frequent vomiting, a coated tongue and fœtid breath, a rapid, weak, or irregular pulse, prostration with, perhaps, tendency to syncope in some cases high excitement and delirium, and in other cases heaviness and stupor. The bowels are more frequently relaxed than constipated, and the stools are generally dark and very offensive. The matters vomited are sometimes nearly black; and the urine, which is often very scanty, and in bad cases almost suppressed, is occasionally sanguinolent. Hæmorrhage from the mouth, stomach, and bowels, or from the respiratory passages, is not an unfrequent accompaniment. In some cases the intellect remains unclouded to the last, while in others the patient dies convulsed or comatose.

To describe at length the different varieties of the Plague, which have been enumerated by authors, would be very unprofitable, and only serve to obscure a subject which has often been made unnecessarily intricate by extreme verbiage in the attempt at over-subtle distinctions. The fever may vary from a simple synochus, or even an urgent synocha with violent delirium, &c., to typhus of a putrid type, with rapid sinking and speedy death. The three forms or varieties of some recent writers appear to be merely three degrees of malignancy, according to the intensity or virulence of the febrific poison, the constitution and condition of the patient, the sanitary state of the locality attacked, and the general sickliness of the season. In an epidemic outbreak the fever is usually much more malignant and deadly at first than at a later period of the invasion, its intractability and fatality very sensibly abating after a period. Sydenham tells us that, "in the infancy of the Plague (in 1665), scarce a day passed but some of those who were attacked died suddenly in the streets, without having had any previous sickness; the purple spots, which denoted immediate death, coming out all over the body; whereas after it had continued for some time, it destroyed none unless a fever and other symptoms had preceded." It seems not improbable that panic has often had a good deal to do with the very rapidly-fatal cases to which the name of 'Peste foudroyante' has been given by French writers. Clot-Bey, in his account of the Plague in Egypt, says that the worst cases usually proved fatal on the second or third day, the cases next in point of severity on the fifth or sixth day, and that in the milder cases death did not generally occur till the second or third week after the first setting in of the symptoms.[1]

During an epidemic, many persons have often been affected with glandular pains and swellings, and occasionally also with carbuncles, but with so little febrile disturbance that they have been able to follow their occupations, and have speedily got quite well under very simple treatment. Such cases have often been the occasion of no little controversy as to whether the persons should be considered as infected with the Plague, and therefore liable to an enforced segregation under the old system of quarantine police. In connexion with this point, it may be noticed that in Egypt and some other countries, where the Plague used to be a frequent visitant, glandular swellings and carbuncular disease are extremely common affections in most seasons.

DIAGNOSIS.—If the presence of bubos, carbuncles, and petechiæ were an invariable and necessary feature of the Plague, and of no other febrile disease, there would, of course, be no greater difficulty in discriminating it than there is in discriminating small-pox or measles from other pyrexiæ. But such is far from being the case. Fevers have repeatedly been alleged in certain countries, and at certain epochs, to be cases of the Plague, although they were at the time unattended with these external phenomena; and, on the other hand, fevers accompanied with these symptoms, occurring in other countries and at other epochs, have as frequently not been designated or considered cases of the true pest. Diemerbroeck, who saw much of the Plague in Holland, during the early part of the seventeenth century, distinctly states that there is no one characteristic or pathognomonic symptom of the Plague; and the remark of Heberden, that, "on first breaking out, the disease has never been known to be the Plague," strictly accords with the observation of all the most experienced writers of the present century, as well as of former times. It is well known that in Constantinople, or in Cairo, no physician ever ventured to say what was the true nature of a prevailing fever, however fatal it might be, or would give it the name of the Plague, until a case occurred in which a distinct bubo or carbuncle was seen. This hesitation was mainly due to the universal unwillingness to admit the presence of a disease, the bare mention of whose name carried with it such dire consequences to the freedom of personal and commercial intercourse; and the result was, that the pestilence had generally existed among a community for a considerable time before any prophylactic or precautionary measures were adopted.

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[1] De la Peste observée en Egypte. Paris, 1840.

That glandular swellings, and occasionally also carbuncles, may be present in other forms of pernicious fever, malarial or not, besides the Plague, has been frequently noticed by writers of different countries. For example, the endemic fevers of the Danubian Principalities, which were so terribly destructive to the Russian armies in the campaign against the Turks in 1828-29, as on all former occasions, and which were called sometimes putrid typhus, and at other times pernicious intermittent, are described as being often accompanied with bubos, carbuncles, and purple blotches on the skin. Their greatest malignancy was in the months of August and September. In the earlier part of the year dysentery, with ordinary intermittents and remittents, were very common and fatal; the latter insensibly lapsed into the pestoid fever. The worst cases were evidently undistinguishable from the Plague; but the authorities studiously avoided all mention of the word, from dread of the panic among the troops that would have inevitably ensued.

In the fever known as the "Pali Plague" in India, to which reference will be subsequently made, the symptoms were often closely akin to, if not identical with, the pestilence of the Levant, &c.; and the same has been the case in some other pernicious fevers, both in India and in other tropical countries. In the endemic typhoid fevers of Syria petechiæ and enlargement of the parotid glands have been noticed as being frequently present, so that it has been difficult at times to distinguish them from the true pestilence; and it is well known that glandular swellings, and even carbuncles, not unfrequently occur in the typhus of our own country. Some striking instances are on record of a fever directly produced by the inhalation of putrescent animal effluvia, exhibiting all the characteristic phenomena of the Plague. In the Medico-Chirurgical Review for January 1825 is related a case of this sort, which occurred to four sailors at Whampoa, who had gone on shore to bury the body of a comrade, who had died of dysentery. On digging the grave, they accidentally opened a coffin which contained a putrid corpse. Two of the men were immediately struck down with the horrible stench, and soon afterwards were attacked with fever, accompanied with petechiæ over the breast and arms; in one of the patients, a bubo formed in the right groin and axilla. Both men died—one on the fourth, the other on the fifth day. On dissection, most of the inguinal and axillary glands were found enlarged and hardened; several of them, when cut into, contained matter. Another man of the party did not sicken with fever until the eighth day after exposure; but for two or three days previously one of the inguinal glands had been swollen and painful. The symptoms were serious for a few days, but eventually the patient recovered. The fourth man was but slightly indisposed.

To make use of so uncertain and variable an attribute as the contagiousness, or the degree of contagiousness, of an existing fever, as a diagnostic mark of the Plague—as some nosologists and other medical writers have done—is obviously illogical, and must inevitably serve to mislead. In the case of the malignant Danubian fevers, several of the Russian medical officers denied their pestilential character, on the sole ground that no distinct proofs of "*contagion par attouchement*" had been observed; while they admitted that all the symptomatic characters of the true Plague were present. Many similar instances might be cited where this fallacious test has been trusted to. Indeed, most of the absurd errors in the history of the disease during the present and last century may be traced to this very source.

MORBID ANATOMY.—The necroscopic appearances observed by Bulard,[1] Clot-Bey, and other French and Italian physicians in Egypt, were in most respects the same as have been noticed in the bodies of patients who have died from malignant congestive fevers, continued or remittent, in other countries, tropical and temperate. The viscera and their investing membranes, whether of the head, chest, or abdomen, exhibited marks of great venous injection, and there was usually more or less serous effusion into the cerebral ventricles, and the cavities of the pleura and peritoneum. All the parenchymatous viscera were loaded with fluid dark blood, and were generally much more lax and softened in texture than in health; the spleen in an especial degree. On the peritoneal covering of these organs, and also of the stomach and intestines, patches of ecchymosis and petechial spots were frequently met with. The mucous surface of the gastro-intestinal canal frequently exhibited the same appearances; and the stomach often contained a quantity of dirty viscid fluid, like a mixture of bile and semi-putrid blood. Some writers have asserted that the mesenteric glands, and indeed the whole lymphatic glandular system, internal as well as external, are always more or less diseased, swollen, discoloured, and often softened, or otherwise altered in structure. Bulard has found the entire chain of glands from the groin to the solar plexus enormously developed, forming a compact mass, to which the veins, arteries, and nerves closely adhered, and imbedded in blood effused into the surrounding cellular texture. Similar appearances have been observed along the course of the axillary glands, when they were chiefly affected. Clot-Bey remarks, that the bubos in the Plague are always formed by swollen lymphatic glands; those in the neck and about the angles of the jaws being independent of the salivary glands, which usually remain unaffected. Even in cases which had proved fatal before the outward appearance of any bubos, some of the lymphatic glands were, he states, almost always found on dissection to be affected; the morbid change varying according to the stage of the disease, from simple enlargement and increased hardness to dark-coloured softening and putrescent degeneration.

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[1] De la Peste orientale d'après les Matériaux recueillés à Alexandrie, à Smyrne, &c. pendant les Années 1833 à 1838. Paris, 1839.

The blood, whether drawn during life or observed only after death, has very generally been found to be darker and more fluid than in health, and only imperfectly coagulating; the clot being loose and pliable, and never exhibiting a true fibrinous or buffy coat, while the serum is often excessive in quantity, and occasionally more or less deeply sanguinolent. After resting for some time, oily globules have been sometimes noticed on the surface, and the whole mass has been observed to pass rapidly into putrefaction.

CURATIVE TREATMENT.—There is little on this head in medical writings at all satisfactory or encouraging in respect of the recovery of the sick, but much that is admonitory as to the baneful effects of an over-active and meddlesome medication, and of neglecting the prime essential in the treatment of all fevers, viz. the inhalation of a pure atmosphere, of equable temperature, at all times, both night and day. Without this indispensable condition, other remedies will be of comparatively little avail.[1] The perusal of the recorded histories of cases of Plague, as observed at Malta in 1813, and in Egypt in 1835, leaves the impression on the mind that the patients would have fared better had they been treated with light nourishing food and cordials frequently administered, together with simple saline or acid medicines, and without active purgation, blood-letting, and such energetic measures. The treatment which is most suitable for ordinary typhus is doubtless that which is applicable to the Plague. In anticipating or in estimating results, it is always most needful to have regard to the period of an epidemic, when the remedies have been employed; otherwise, the most misleading mistakes may be fallen into respecting the value of remedies or modes of treatment. I cannot better close these few remarks on this head than by quoting the words of a recent experienced writer,[1] in regard of the treatment of other bad forms of fever, viz. the pernicious remittent and yellow fevers of tropical climates.

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[1] Nowhere have the effects of crowding the sick, and of the neglect of hygienic measures in fever, been so dreadful as in the pest hospitals or lazarettos, even within the last thirty years. Dr. Bulard said of the hospital at Smyrna, "Il n'est que le vestibule du sépulcre;" and it was doubly, literally true, for the cemetery was within the walls of the establishment. In the British colony of Malta matters were no better in 1813; few of the patients sent to the lazaret left it alive. Of twenty-eight inmates seen by Sir B. Faulkner on one occasion, and some of whom then seemed to have not been seriously ill, all perished within forty-eight hours; the only attendants were convicts! See his Treatise on the Plague, from facts collected during the author's residence in Malta. London, 1820.

"In considering this subject, it should ever be kept in mind that not only in different situations and countries, but also in different years, these diseases, whatever the form of fever, may vary more or less, and if not in type and character, at least in intensity and complications; so that the remedial means which may have been found useful in one epidemic may fail in another, each, it may be, having a constitution of its own. We are told by Sydenham how difficult he found it, on the breaking out of an epidemic, to determine on the best mode of practice to be pursued, and how he came to a decision only after *ingenti adhibita cautela, intentisque animi nervis*—an example, this, well deserving to be followed."

NATURAL HISTORY, CAUSATION, PROPHYLAXIS, &c.—Prior to the end of the seventeenth century, the Plague seems to have been as truly endemic—with occasional outbursts of epidemic violence—in most of the countries of Europe, including our own, as it was in the Levant and in Egypt from remote times, and continued to be during the first forty years of the nineteenth century. In London, for example, during the first seventy years of the century, not a year passed without some deaths from the Plague being registered; and epidemics occurred in 1603, 1625, 1636, and 1665. The last, known as the Great Plague, was followed by a marked decline in the prevalence of the fever; the number of deaths from it became fewer and fewer, and after 1679 none have been recorded in the bills of mortality of the metropolis. Whether it continued to linger in other parts of England after this date, I am unable to say; for it is to be remembered that the disease had not been limited to London, but was widely spread over different parts of the kingdom, just as typhus is at the present day. In Holland, too, the decline and disappearance of the pestilence seem to have taken place about the same time as in England, or somewhat later.

During the eighteenth century, although there was a marked diminution in the persistency of the disease in a sporadic form, and in the frequency of occasional wide-spread outbursts throughout Europe generally, many severe and very fatal epidemics occurred in different countries, as in Poland, including Dantzic, and other ports in the Baltic, in 1710; in Provence and other parts of Southern France, and especially Marseilles, in 1720-21; at Rochefort in 1741; at Messina and other towns in Sicily in 1743; in several districts of Portugal in 1757; in Wallachia, Podolio, &c. in 1770; and at Moscow in 1771.

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[1] On Diseases of the Army, by Dr. J. Davy, F.R.S. 1863.

In the present century the chief seats of the pestilence have been in Egypt, Syria, Asia Minor, and the coast of Barbary. For details on this point I would refer to a "Sketch of the Geography, &c. of the Plague," in the Brit. and For. Med.-Chirurg. Rev. for April 1864.

The most recent recognised appearance of the pestilence occurred in the neighbourhood of Benghazi, between Alexandria and Tripoli, on the African coast, in 1858. It has been described by Dr. Bartoletti, who was sent by the Turkish Government to investigate its history, in a memoir addressed to the Imperial Society of Medicine of Constantinople, in August of that year, and of which an abstract is given in the Quarantine Parliamentary papers afterwards referred to. A brief notice of the "Pali Plague" of India may be here introduced.

This pestoid fever was first recognised in Cutch, in the summer of 1815, after a season of great scarcity and distress. From that year to 1820 it prevailed in different places in Guzarat, spreading to Scinde in a N.W. direction, and also towards Ahmedabad and other places in the British possessions eastward. The fever was remittent in character, with a great tendency to become continued, of a very adynamic type, and extremely fatal. In most cases there were glandular swellings in the groins, axillæ, and neck. Carbuncles or petechiæ are not mentioned as being present. There was often dyspnœa with cough and bloody expectoration. Vomiting, of at first bilious matter, and subsequently of a dark, coffee-coloured fluid, was likewise a not unfrequent symptom. In some cases the urine was sanguinolent, and blood oozed from the gums. After the beginning of 1821, there was no recurrence of the fever known until 1836, when it was observed in the town of Pali (lat. 26° N. and long. 74° E.), then the principal depôt of traffic between the coast and the N.W. Provinces of India. It spread to numerous places in Marwar in that year, and in 1837 on to the middle of 1838. In 1849 there was a similar fever in Gurwah and Kumaon, on the southern slopes of the Himalayas, and in 1853 in Rohilcund. As to the nature and affinities of the fever, Dr. Morehead, in his valuable Clinical Researches on Disease in India, remarks:—"The description of jail or hospital fever by Pringle, in his work on the Diseases of the Army, has considerable resemblance to that of the fever at Pali. . . . The causes were supposed to be crowding, filth, and effluvia from decomposing animal and vegetable matters."—*Second Edition*, p. 158.

In every country where the Plague (and pestoid fevers in general) has prevailed, certain local conditions have been found to favour its *development and spread.* "The principal of these are, residence upon marshy alluvial soils along the Mediterranean, or near certain rivers, as the Nile, Euphrates, and Danube; the dwellings of the people being low, crowded, and badly ventilated; a warm, moist atmosphere; the action of putrescent animal and vegetable matters; insufficient and unwholesome food; and physical and moral wretchedness."[1] An elevated site, even in the immediate neighbourhood of an infected city, has often remained quite exempt, although intercommunication was not interrupted—*e. g.* the citadel of Cairo, and the village of Alem-Daghe, near to Constantinople. The higher parts of Valetta suffered very little in the Malta epidemic of 1813; the ratio of the attacked became greater and greater in descending from the higher to the lower levels of the city, towards the foul shores of the harbour; and so much less frequently were the occupants of the upper and more airy storeys of the lofty houses attacked than those of the basement floors, that it was a common remark, Dr. Hennen (in his Medical Topography of the Mediterranean) says, that "the Plague was a disease which seldom went upstairs." It has been very generally in the crowded and filthy parts of a town that the earliest cases of an epidemic have occurred, and the chief sufferers have been invariably the poor and neglected. The state of most Turkish or Egyptian towns in the present age represents very nearly what London and many other European cities were in the seventeenth century.

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[1] Rapport à l'Académie Royale de Médecine, sur la Peste et les Quarantaines, fait, au nom d'une Commission, par Dr. Prus. Paris, 1846.

Outbreaks of the Plague, as of typhus, have often followed in the wake of famines and other desolating calamities. This was strikingly the case with the visitation at Benghazi. For two or three years previously there had been an unusual drought, and the cattle had perished in great numbers from an epidemic disease. In 1857 the destitution of the Bedouin tribes became extreme, and it was then that the pestilence commenced. "Why it was the Plague rather than typhus, I know not," says Dr. Bartoletti; "but the fact was so; and I may add that the great epidemic of Plague at Erzeroum, in 1841, was also preceded by a terrible famine. One of the essential conditions for the production of typhus was wanting, viz. the agglomeration of human beings in a confined space."

Epidemic Plague has generally been preceded by a sickly season, —the sickliness consisting in the great prevalence and severity of the ordinary endemic fevers, of fluxes and other forms of bowel complaints, and not unfrequently also of catarrh. Sydenham's account of the epidemic constitution, previous to the great Plague of London, may be taken as typical of what usually occurred in respect of the disease during the sixteenth and seventeenth centuries in this and in other countries of Europe. In the spring of 1665 catarrhs and pulmonic disorders were very prevalent and fatal. About the same time a bad form of fever, attended with vomiting, diarrhœa, &c., began to be very common, and this was increased in severity as the season advanced. Towards Midsummer, cases of this fever (which Sydenham calls "pestilential,") were accompanied with bubos and carbuncles on the surface: then only was it recognised and designated as the Plague. The pestilence went on increasing in deadliness until the third week in September, when nearly 8,000 died in the course of the week, although two-thirds of the inhabitants had by this time fled from the city. It had then reached its acme, and forthwith began to abate. It very nearly ceased on the approach of cold weather; a few sporadic cases only occurred during the winter and following spring. The same form of fever, however, as had preceded the first recognised cases of the Plague, was again observed throughout 1666; but it was not so general as in the previous year. Sydenham expressly says that it was of the same species as the Plague, only not so violent—*revera enim cum ipsissima Peste specie convenit, nec ab eâ nisi ob gradum remissiorem discriminatur.*

Morton, a contemporary of Sydenham, has remarked of the Plague, as he saw it in London, that "it often appeared under the form of a continued or remittent fever; but this changed into the other, and *vice versâ;* and that each in its turn became epidemic, the one yielding to the other." A similar remark has been made by many other writers in former, as well as in more recent, times. The epidemics in Egypt, in 1835 and 1841, were ushered in by the unusual prevalence of the ordinary endemic fevers, in some places of a continued, and in other places of a periodic, type.

The influence of *season* on the prevalence of epidemic Plague will be seen from the following facts. In England the pestilence was most severe in the epidemic years 1603, 1625, 1636, and 1665, from about the middle of July to the first or second week in October. The Plague at Marseilles raged most fatally in the autumn months; and the same was the case at Moscow in the epidemic of 1771, when upwards of 1,200 deaths took place for several days out of a population of 150,000. In Constantinople it has generally reached its acme in September; in Smyrna about a month sooner; and the same may be said of Tunis, Algiers, and other places on the North African coast, where it has usually manifested itself in spring, and committed its greatest ravages in July and August. Malta suffered most in June and July. In Syria the summer months have ordinarily been the most fatal. In Egypt most epidemics have commenced towards the end of the year, and gradually advanced, reaching their acme in March or April, when the southerly winds are most prevalent. The disease generally ceased in the second or third week of June. At Cairo it never continued, it has been said, beyond St. John's Day, 24th June. It thus seems that in Egypt the chief prevalence has generally been a good deal earlier than in other countries. Volney remarked on this subject:—"The winter stops the Plague at Constantinople, because the cold is great; and the summer lights it up, because the heat is then humid: while in Egypt the winter favours it, because the climate is then warm and moist; and the summer stops it, because it is hot and dry. The heat is only injurious when associated with humidity." That the peculiar meteorology of the climate of Lower Egypt may have something to do with the point in question, seems very probable.

Mention is often made, in the history of Plague epidemics, of the weather having been before, and during, their prevalence remarkably oppressive and distempered. This was notably the case, according to Maitland in his History of London, in the Great Plague, when, for months, scarcely a breath of air was to be felt, and the little that there was came from the south; it was generally hot and stagnant, and mildews were abundant. A like state of atmosphere has often been noticed in other countries. Dr. Brayer specially alludes to the circumstance in his account of the Plague in Constantinople. Dr. Hennen, in his narrative of the visitation at Malta, relates the fact, as indicative of a distempered atmosphere, that, during the prevalence of the fever, "every whitloe festered, and every scratch became an ugly sore; a tight shoe was sufficient to produce a livid boil. The military hospitals were crowded with such cases."

The mode in which the pestilence has been wont to manifest itself in a place, and to spread among a community, may be gathered from what has been said above respecting the usual antecedent state of the public health, and the physical conditions, &c. of the localities chiefly attacked. It has been often alleged that outbreaks of the Plague have been known to occur among a population previously quite healthy, immediately or very soon after the arrival or introduction *ab extra* of a person or object already affected with the disease, or believed to be impregnated with its infective poison; and that from the first case or cases, as from a focus or centre, the subsequent extension could be distinctly traced. But whenever there has been an opportunity of ascertaining the real and complete history of the facts, the course of events has been found to be much less simple and consecutive. Unfortunately, most of the past history of Plague epidemics rests on mere rumour, or on most imperfect information. It has only been within the last thirty years that thoroughly trustworthy records on the subject have been published. The admirable Report of the French Academy has shown that the pestilence has generally appeared in single cases or small groups of cases, at or about the same time, in different spots or localities of a town, or perhaps in different districts of a region, often considerably distant from each other, and without any traceable direct communication between the persons attacked. Sometimes several towns have become the seats of the fever nearly simultaneously, the intermediate villages remaining exempt, while at other times it has advanced in a more regularly progressive manner, invading a number of places "*de proche en proche*," and in succession, over an extensive tract of country. That the Plague may be diffused by transmission from the sick to the healthy near to them—in other words, by personal *contagion*—cannot be reasonably doubted; and it is equally certain that both its development and spread have often occurred independently of such agency. Extravagant opinions on this subject were in vogue among most physicians until of recent years, when the accurate observation of facts and a rigorous examination of previously recorded testimony sufficed to show that the influence of contagion in the dissemination of the Plague is very much the same as in the dissemination of the bad forms of typhus, and that the laws which regulate its activity and power in the one disease are equally true in respect of the other.

The limits of the present paper preclude any discussion of the various important questions of State Medicine connected with this subject, and which relate to the public measures to be adopted for the prevention or arrest of the Plague, and for the protection of countries from its introduction by shipping and other channels of intercourse. To such measures the term of *Quarantine* has been usually applied. For full information on the subject I would refer to the Report of the French Academy,[1] and to the elaborate documentary evidence, and report thereon, prepared by a committee of the National Association for the Promotion of Social Science, and printed, by order of the House of Commons, in May and August 1860, and August 1861.

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[1] A summary of this Report, with Introductory Observations, Extracts from Parliamentary Correspondence, and Notes, was published by the writer of this article in 1846.

ERYSIPELAS.

By J. Russell Reynolds, M.D. F.R.S.

Definition.—An acute specific disease characterised by fever of a low type and a peculiar inflammation of the skin. This inflammation exhibits a marked tendency to spread over the surface, to induce serous infiltration and suppuration of the areolar tissue in its neighbourhood, to affect the lymphatic vessels and lymphatic glands, and to cause serous exudation between the cutis and the cuticle.

Synonyms.—*Scientific Names.—ἐρυσίπελας* (Greek); Febris erysipelatosa (Sydenham); F. erysipelacea (Hoffmann); Rosa (Sennert); die erysipelatöse Dermatitis (German); Erysipèle (France); Risipola (Italian).

Popular Names.—Ignis sacer (Latin); die Rose, der Rothlauf (German); the Rose (Scotland); Saint Anthony's fire (England).

Natural History.—Causes.—Local irritations of the skin, such as the application of blistering fluids, or of boiling water, produce dermatitis, but they do not, of necessity, cause Erysipelas. The inflammation they produce may be very severe, and may resemble that disease; but the differences between such common inflammation and the special malady we are now describing are greater in number and more important in kind than are their points of resemblance. There is, then, some "cause" of Erysipelas over and above that, whatever it may be, which produces simple inflammation of the skin. There are some facts which go far to show that this cause, or that some one or more of a number of concurrent causes, may exist in the "individual;" for it is well known that some persons are liable to suffer from repeated attacks of Erysipelas, and these either with or without the slightest provocation; whereas others may be blistered, burned, cut, torn, or otherwise injured, without exhibiting any indication of the existence of such tendency. On the other hand, it is equally clear that Erysipelas sometimes has an "epidemic" character; and it is still more common to find it haunting certain localities, and thus exhibiting the features that we term "endemic;" so that in these cases its most effective cause would seem to be outside the individual, viz. in some external circumstances. If we admit, as indeed we must do, the special liability of some individuals or families to the occurrence of this disease, then the conditions underlying such liability must be regarded as "predisposing causes" of Erysipelas, and we must seek still further for the so-called "exciting causes" of the affection; and these may commonly, but not invariably, be found in accidental or other injuries to the skin, such as exposure to cold or heat, to moisture, or physical abrasion. If we accept, on the other hand, the presence of a distinct morbific agent—either epidemic or endemic—as the efficient cause of Erysipelas, then the constitutional state of the sufferer sinks into comparative unimportance, although we may still retain some belief in its action as a predisponent.

The truth as to causation lies, most probably, not between these two ideas, but in their combination; and such conclusion is by no means at variance with the belief that sometimes the one and sometimes the other factor is the more influential. It may be that either one may sometimes be so potent as, *per se*, to produce the disease; but it is more probable that, in all cases, there is some constitutional predisposition, and also some agent operating from without.

Among constitutional predisponents the most important is that of which we know nothing more than the fact of its existence, viz. an individual or family proclivity to the disease. The next in value is age. Erysipelas is common in newly-born infants, but rarely occurs between the first year of life and the twentieth; after this period it is frequent, as an acute affection, till the fortieth year; whereas in more advanced age it is seldom seen except as a chronic, or sub-acute, and less important malady. It has been stated that women are much more liable than men, and especially so during menstruation, and at the climacteric period; but such statement is not supported by reliable facts, although a woman, subject to Erysipelas, may exhibit the disease with especial frequency during the catamenial flow. Little that is of any value can be said of "temperament" as a predisponent; but it appears probable that the "gouty diathesis" increases the frequency of its occurrence.

In addition to the "exciting" causes already mentioned—viz. all undue impressions upon the skin—we must enumerate errors in diet, and especially the taking of certain things, such as shell-fish, or improperly smoked, dried, salted, or otherwise "half-preserved" meats. But by far the most important cause, acting from without, is the "poison," whatever may be its nature, which exists in one case and can be communicated to another, either by inoculation, simple contact, transmission through the air, or by fomites.

It is not intended in this work to deal with Erysipelas as it is commonly seen in the surgical wards of hospitals, but with that form of it which, arising often without any distinctly defined external cause, and certainly in the absence of any proved contagion, has a history differing widely from that of the disease as it is seen to follow surgical operations, under circumstances of apparently endemic aggravation. It is not asserted that the disease is essentially different in the two classes of cases; but it is held that their clinical history is so different, that it is desirable to treat them separately, and to confine the description given in this "System of Medicine" to the latter, which usually falls under the care of the physician.

Symptoms.—These are both general and local: on the one hand there is fever, on the other definite structural change in the skin, mucous membrane, and, it may be, in the subcutaneous and submucous tissues. When fever precedes the appearance of redness on the skin, the disease resembles one of the exanthemata: when local inflammation exists before marked pyrexial change, the case, in its general career, is more like that of pneumonia, pleuritis, or inflammation of any other organ. The former course is the more common of the two.

The *commencement* of Erysipelas is usually marked by uneasiness of not very definite character; rigors, slight shivering, or only a feeling of chilliness may mark the onset of the malady; but more commonly rigors do not exist at the beginning, they occur after several hours of discomfort, and either immediately before or simultaneously with the appearance of local inflammation. Usually, then, prior to the occurrence of rigors, there is malaise, aching of the limbs, loss of appetite, thirst, nausea, or vomiting, diarrhœa, soreness of the throat, increased heat of skin, and frequency of pulse, headache, giddiness, confusion of thought, feeling of depression in spirits, epistaxis,—as an occasional event,—and, in fact, all the signs of pyrexial disturbance, without any such special predominance of any one of them as should enable the physician to forecast the nature of the impending evil.

It is not possible to affix a definite duration to these symptoms: sometimes they commence after, sometimes simultaneously with, the local changes; but more commonly they precede the latter by a few hours, *i.e.* from eight to ten, or by two or even three days. It often happens that a man feels well in the morning and at mid-day, but towards evening is uneasy; passes a restless night, growing worse from hour to hour; and on the morning of the next day observes some redness of his nose or ear. Or feeling better, but not well, on the second morning, he goes through a day of increasing discomfort, which becomes very considerable towards evening; passes a second night worse than the first, sometimes accompanied by delirium; and the special phenomena of Erysipelas appear on the third day. But their appearance may be delayed until the fourth or even fifth day from the onset of symptoms.

On the appearance of the cutaneous inflammation there is no remission of the pyrexial symptoms; on the contrary, they are sometimes aggravated in intensity. The usual site for their development is some part of the head; but they may appear in any other portion of the body. Local disease or injury of the skin, or even of the subcutaneous tissues, may determine the place of commencement. Abrasions, scratches, or wounds, wherever they are situated, may be the starting places of specific inflammation of the skin; whether this be of the kind we call Erysipelas, measles, or scarlet fever. But this determining power is not limited to such injuries or affections as "break the skin;"

for the locality of appearance, and even recurrence, of Erysipelas may be determined by the presence of gout in a particular joint of either the upper or lower extremity, and also by the irritation of diseased teeth in either the upper or lower jaw.

Usually, however, the nose or the ear is the point at which the inflammation may first be seen. The change is, so far as my own observations extend, commonly seen to commence in close proximity to one of the passages through the skin, *i.e.* where the skin undergoes that transition which consists in its becoming what we term mucous membrane. Thus, not only at the nose and at the ear does Erysipelas begin, but it commences just where the skin of the nose turns upwards into the nostril, or just at the point where the skin of the ear loses the dryness and other characteristics of ordinary skin. Again, Erysipelas often is noticed first at the angles of the mouth, or at the edges of the eyelids; it is met with at the anus, about the genital organs, and in the neighbourhood of the recently-divided umbilical cord.

To the patient the part affected feels hot and irritable; and, upon touching it, sore, stinging, and smarting. It is of red colour and shining aspect; it is warmer and harder than the surrounding tissues, swollen, and, as the disease advances, very tender to the touch. The inflammation extends from the spot first affected, sometimes in all directions, but more commonly in one much more rapidly and more widely than in another. For instance, Erysipelas starting from the ear will sometimes extend downwards and not upwards, backwards and not forwards, and so on; whereas in other but, I believe, rarer cases the progression appears to radiate about equally in all directions. At the advancing edge of Erysipelas the elevation of skin (swelling) may often be not only felt but seen, and that most distinctly; whereas at the receding margin there is so gradual a decline of swelling that it would be difficult to say with certainty that it existed. Where the inflammation is advancing the line is marked not only by elevation of the surface, but by a sharply-defined difference of colour; the white or pale healthy skin is invaded by a distinct line of red, with an occasional streak, branched or not, in advance of the general boundary. Where the skin-affection is receding there is no such abrupt transition, but the heightened tint of the most active inflammation is gradually shaded down through medium and mixed tints until, without any clear line of demarcation, the skin is found in its ordinary healthy state.

Sometimes the amount of swelling is not considerable; at other times it is enormous; and the disfigurement is such that none would recognise the features of the sufferer, nor for a moment think that they were features at all, or even parts of any human being. The amount of swelling is greatest where the skin is the most loosely attached to the subjacent structures, and where there is much areolar tissue which can be distended with fluid. Thus we find the eyelids and the neighbourhood of the mouth the most disfigured in appearance, and all trace of the former may be completely obliterated by the effusion. The swelling, when confined to the skin, is moderate in amount, uniform in elevation, hard to the touch, pitting only slightly on pressure, and shading off on the side of recession, but terminating more or less abruptly on that of advance. When the areolar tissue is much infiltrated, the swelling is carried to a higher degree, its surface is irregular in elevation and consistence, and there is often deep pitting upon pressure. Under the latter circumstances there is generally suppuration, probably determined by the sloughing of small or larger portions of the subcutaneous areolar tissue.

The surface of the inflamed skin remains, in mild cases, intact throughout, exhibiting, besides redness, only slight increase of desquamation as the malady dies away. In more severe and more common cases there is some vesication of the surface; little bladders are seen like those produced by a blistering fluid, or a scald. Whereas, in very severe cases, large bullæ, of irregular shape, make their appearance; they soon burst, and leave dry and thick crusts, which render still more hideous the face that they have covered. It often happens that the inflammation is extending in one direction and receding in another, so that Erysipelas in all its stages may be witnessed at the same time in the same individual; but the maximum of redness and of swelling is usually reached on the second or third day.

In almost all cases there is distinct inflammation of the neighbouring lymphatic vessels, with pain, swelling, and tenderness of the lymphatic glands: but suppuration of the latter is not met with in ordinary cases, although severe. Inflammation of the lymphatics is most commonly observed when Erysipelas has been set up by a poisoned wound.[1]

The pulse is generally full and with a frequency varying from 100 to 120 in the minute; the heat of skin is well marked, perhaps over the inflamed surface unduly marked in comparison with the elevation of the temperature generally. This may be owing to the local arrest or diminution of transudation and evaporation.

In a severe case, recently under my care, the temperature in the morning of the eighth day of illness, but fifth day of eruption, was 104° Fahr.; on the next day $102\frac{3}{5}$°; on the following $103\frac{1}{5}$°; then came twenty-four hours in which it ranged between $99\frac{3}{5}$° and $100\frac{1}{5}$°; to be followed by another rise,—coincident with some extension of the inflammation,—to $102\frac{3}{5}$°; after which it became normal. The evening temperature in this case was daily lower, and sometimes considerably lower (2° to 4° and even 5°), than that of the morning. A similar relation between the morning and evening temperature was observed in another almost equally severe case occurring at the same time. The prevailing relation between morning and evening temperatures is, however, similar to that observed in other acute specific diseases, viz. an excess of elevation in the evening; but the variation from this general type is, I believe, more common than is supposed not only in Erysipelas but in other allied maladies. It is quite clear that relapses are attended by renewed rise of the thermometer; and such relapses may occur in the morning as well as in the evening, and so pervert the characteristic febrile course. Such relapses may possibly be overlooked, as in some cases the symptoms are almost imperceptible to the patient, and may occur in some locality which is not necessarily exposed to the eye of the physician; and yet, although so slight as not to attract attention in any other way, they may do so by their effect on the thermometer. Lately, for example, in a patient apparently convalescent,—feeling tolerably well, and taking food with relish, the temperature having been normal for two days,—I have seen a sudden rise in the thermometer, unattended by any return of pain or malaise, but, upon examination, shown to be coincident with renewal of the inflammation below the shoulder blades; it having commenced at the ears, extended to the back of the neck, and for some days stopped at the level of the scapular spines.

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[1] Niemeyer, Lehrbuch der speciellen Pathologie und Therapie. Bd. ii. p. 396.

The fever, as measured by the thermometer, is very variable in *duration;* and the temperature, after having returned to the normal amount, may exhibit several re-elevations coincident with extensions of the inflammation. Usually the maximum is reached on the third day of eruption, and the decline commences on the fifth or sixth.

The pulse,—usually exhibiting an increased frequency bearing direct relation to the abnormal elevation of temperature,—may revert to the habitual standard at the end of the third or fourth day, and not again rise far above this, although one or more relapses may occur, each of which is marked by a rise in the thermometer.

Albumen appears in the urine in many cases. It may make its appearance from the fourth to the eighth day, or even later, in relapse. Unless there be pre-existent disease of the kidneys, it is small in amount and of short duration; it has been noticed to be absent on the fifth day, present on the sixth, and again absent on the seventh. The quantity of urea is increased, while that of the chlorides is diminished.

The *course* of symptoms varies widely. In one class of cases there is a speedy diminution of their severity both locally and generally; whereas in others the reverse is observed. Sundry local changes, proportionate to the amount of swelling, may occur, and become excessively annoying. Such, for example, are blindness, deafness, and impossibility of breathing through the nose. But, beyond these and the relapses which have been already mentioned, there are others depending upon the sloughing and suppuration of areolar tissue. When such changes take place, the symptoms become much more distinctly adynamic; and in bad cases the tongue is brown, the lips and gums are covered with sordes, the pulse rises in frequency and loses in force, so that it is often quite uncountable; there is low muttering delirium, with jerking contractions of the limbs, and, indeed, all the other signs of impending dissolution.

DIAGNOSIS.—It is not very easy to confound Erysipelas with any other malady when once its ordinary symptoms are developed; but it may sometimes be inferred to be present before the skin has shown signs of inflammation. Where there is marked pyrexia, with vomiting at its commencement, without notable pain in the back, or obvious change in the mucous membrane of the throat or nose, but with enlargement, pain, and tenderness of the lymphatic glands in the neck, Erysipelas may be, and ought to be, suspected. Attention to the description of symptoms already given will be sufficient to prevent any errors of diagnosis. The spreading character of the inflammation, as this is seen in the skin and also in the subcutaneous cellular tissue, distinguishes Erysipelas from true phlegmon, where lymph speedily circumscribes the swelling. Although the symptoms of Erysipelas occasionally exhibit intermissions, and by no means rarely some remissions, it would be difficult now for the mistake between it and intermittent fever to recur.

From what has been termed "diffuse cellular inflammation," Erysipelas may be distinguished by the constant presence of inflammation of the skin, which latter bears a direct ratio to the affection of the areolar tissue, and precedes it, as a rule, in regard to time.

Erythema differs from Erysipelas in the comparative mildness of its general symptoms, in the absence of swelling of the skin, and in the tendency which it exhibits to form patches, of various sizes and shapes, which show no marked propensity to spread.

PATHOLOGY.—There can be no doubt that some cases of Erysipelas resemble those of simple erythema, or simple inflammatory redness of the skin, such as may be produced by a mustard poultice. Nor can there be any doubt that, on the other hand, there are cases of Erysipelas which resemble more closely diffuse cellulitis and pyæmia. Between these extremes there are patients whose symptoms are those of phlebitis, or of inflamed absorbents; and others whose cases can hardly be distinguished from genuine phlegmon. In all these we may have no doubt of the existence of Erysipelas; but the idea we entertain of the "pathology" of this disease will be determined by the frequency with which we find it assuming this or that typical form, the amount of importance we attach to these several associated conditions, and the clearness of our knowledge about them. Thus, Erysipelas may be regarded as a disease having its first local manifestation in the absorbent system, or it may be held to be essentially a blood disease, always called into play by some external injury, however slight that injury may be; it may be thought to be so distinctly hæmic, that it should find its place among the symptoms of pyæmia; while, on the other hand, the view may be entertained that a true inflammation of the cutis is its one essential condition,—that Erysipelas is a *morbus per se*, having powers of easy association with each of those maladies alluded to, but being, at the same time, essentially distinct from all of them. Those who entertain the last opinion, again differ among themselves, some regarding the inflammation as identical in its essence with that which might be produced by any irritant; others, as being of special type, the result of one specific poison, the presence of which in the system is the *sine qua non* of the existence of that form of inflammation which we term Erysipelas. And here, in this last resort, diversity of opinion may still be found; for some hold that the poison is developed from within, that it arises from "crudities," from "digestive derangements," and the like; while others believe that it is always imported into the body from without; and it would seem that yet a third view might be taken, viz. that it is by the conjunction of these two elements that the disease is established; that is to say, neither internal conditions (predisponents), existing alone, nor external poisons (septics, epidemic or endemic influences), acting by themselves, can produce Erysipelas; but that the outside poison, however active, or however greedily swallowed by the organism it may be, is inoperative unless it find in that organism the proper, *i.e.* "special," nidus, conditions, or material for its development.

It would be possible so to select and arrange the facts about Erysipelas as to make them support any one of these theories or pathological positions; but, taking them without selection, and only arranging them so far as to render their teaching intelligible, the conclusion most consistent with them appears to me to be that which is expressed the last in the foregoing paragraphs, and in the definition of the disease; viz. that Erysipelas is an inflammation of the skin, that it may involve the absorbent or the venous systems, that it may change the character of the blood; but that it may exist independently of any such complication; further, that it is an inflammation of "special" character; and, lastly, that it depends upon the action of a particular poison upon a peculiarly predisposed constitution.

MORBID ANATOMY.—The slighter cases of this disease rarely furnish opportunities for the study of their anatomical conditions, but analogous states of the skin and neighbouring tissues may be observed in the outskirts of the severest inflammation in cases of fatal Erysipelas. In them, after death, as well as during life, the disease may sometimes, but of course at different points, be observed in all its stages, and in all its degrees of intensity.

The skin is thickened by increase of vascularity and of serous infiltration, while there is a marked absence of lymph so thrown out as to circumscribe the swelling. In mild cases, no suppuration is observed, but, in the less mild, there are detached patches of suppuration, sometimes affecting the cutis only, but more commonly extending into the areolar tissue; whereas, in severer cases, the skin may be completely separated from the subjacent tissues, and this for a very considerable extent, by large quantities of pus, in which shreds of sloughing or dead areolar tissue may be found, semi-detached or floating. The disease, although commonly limited to the skin and its immediately connected tissues, sometimes extends to the deeper and more important structures, and then muscles and ligaments and bones become involved in the general mischief.

Mr. C. de Morgan[1] states that a very important fact had been "mentioned" to him by Mr. Busk, viz. "that in all the fatal cases which he examined, the lungs were highly congested, and that, on close inspection, the smaller pulmonary vessels were always found to contain pus; that, in fact, a minor degree of pyæmia was always present. He (Mr. Busk) has observed the same thing in the small vessels of the head, when that part has been the seat of Erysipelas." Dr. Bastian has found minute embolic masses in the small arteries and capillaries of the grey matter of the cerebral convolutions. Some of these masses, which are small, appear to be made up of white blood-corpuscles; but the larger, irregular, and rounded bodies are, in Dr. Bastian's opinion, amorphous masses of albuminoid material, separated from the blood-plasma.[2] Dr. Copland[3] says, "The veins proceeding from the part chiefly affected are often inflamed, or contain pus, as first observed by M. Ribes, and confirmed by Messrs. Dance, Arnott, and by my own observations." It is well known that in many cases of fatal Erysipelas evidences of disease may be found in the spleen, liver, kidneys, lungs, bronchi, larynx, trachea, and fauces; but there is nothing specific in the character of the changes discovered in these organs—nothing, that is, which is peculiar to the disease called Erysipelas—nothing, indeed, which depends upon the Erysipelas *per se;* but all that may be found is only the sign of such general blood-change as may be associated not only with the disease now under consideration, but also with that large group of maladies which stand in close relation with pyæmia. (See p. 553.)

Occasionally, gangrene is found in some portions of the inflamed skin or cellular tissues; and this particular termination of Erysipelas has appeared to me the most common in those cases which are associated with dropsical effusions. Sometimes the Erysipelas has appeared around natural or artificial openings made for the escape of serum, and then either portions of the integuments of the lower extremities, or, more commonly, of the genital organs, become greatly inflamed and gangrenous. The association of gangrene with dropsy may be due, in some measure, to the altered blood-condition—which almost always exists in extensive anasarca, whatever may be the seat of that mechanical obstruction to which it is referred, and which determines the locality of its appearance,—an altered blood-state, moreover, which in some dropsies appears to constitute the whole of the essential condition for their development. But, in addition to the blood-change, there is, in the fact of the existence of dropsical effusion in the cellular tissue, a change in the nutrition of the vessels, and an altered relation of the fluids and solids of the parts involved; and there is, further, a simply mechanical interference with the circulation in the skin, exerted by the pressure of the effused fluid not only on the capillaries, but on the vessels which lead to and from them. Thus, dropsical limbs are, unless reddened by Erysipelas or other conditions not essential to the dropsy, ill-nourished, pale, and cold; and a slight addition of difficulty to the already embarrassed circulation of the part, often ends in gangrene.

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[1] Holmes' Surgery, vol. i. p. 237.

[2] British Medical Journal, January 23, 1869.

[3] Dictionary, art. "Erysipelas."

Prognosis.—A case of Erysipelas is bad in proportion to the predominance of the symptoms of blood-poisoning over those of simple inflammation. It is bad just in the degree to which it resembles typhoid fever or pyæmia, rather than simple dermatitis. Mere extent of inflammation is not of itself of evil augury; a high degree of inflammation is of no greater value; but a very rapid and weak pulse, with a dry brown tongue, and low muttering delirium, with marked prostration of the strength, is of almost fatal omen, although the local changes may be closely limited in both distribution and severity.

Cases which arise from the introduction of poisonous matter are worse than those in which the malady appears spontaneously; and this, whether the poison has been introduced by a wound made with an infected implement, such as a dissection wound, the prick by a bone from diseased meat, &c.; or whether it has been conveyed through the air, or by other means, to a wounded surface, placed where Erysipelas is endemic.

The extremes of age, the presence of disease in either the kidneys or the liver, the dropsical constitution, a state of chronic alcoholism, or of any morbid blood-condition, are prognostic of an unfavourable termination.

The extension of Erysipelas to the throat may introduce a source of danger altogether different from that which belongs to the disease itself. Life may be threatened and indeed terminated, and that too very suddenly, by apnœa. The inflammation of the fauces may bring about serous or other infiltration of the neighbouring submucous tissues, and the opening into the windpipe may be closed. In Erysipelas of the head or neck it will always, therefore, be necessary to examine the throat most carefully, and to observe the manner and number of respirations, as well as the tint of the skin, in order to guard the prognosis against a false security. It is the more necessary to do this, because in some cases the patient—owing probably to the dulled state of his sensations and perceptions, brought about by the poisoned condition of his blood—may make no complaint of dyspnœa, or of discomfort in the throat; whereas an altered tone of voice or cough, an occasionally hurried respiration, a slight lividity of the lips or finger-nails, an undue movement of the alæ nasi, or any other signs of impaired respiration, may lead to the discovery of danger the most grave and imminent.

Again, the appearance of symptoms of disturbance in the nervous centres, over and above, or out of all proportion to that which might be accounted for by the general febrile condition, is of very serious omen. The occurrence of delirium, and especially of nocturnal delirium, is of comparatively little importance; but a marked drowsiness —sometimes alternating with delirium, sometimes persisting and increasing in intensity—is a very serious symptom, forecasting that mode of termination which is by no means rare in Erysipelas, viz. "coma" from either effusion within the cranium or impairment of the brain-nutrition by embolism of the small vessels. It is, however, by no means probable that in all cases terminating thus, there is or has been an extension or metastasis of inflammation to the membranes of the brain. In some instances there are symptoms of "meningitis" observed during life, and evidences of its presence and its results may be discovered after death; but in others the cerebral symptoms are those of oppression rather than excitement, and the post-mortem appearances are those of effusion only, or of that embolic occlusion of vessels described by Dr. Bastian. In the latter class of cases the symptoms are probably due partly to the direct effect of altered blood upon the nervous centres, and partly also to the indirect effects of that alteration in leading to passive effusion, or obstructed circulation. The relation between dropsy and Erysipelas has already elicited remark.

Erysipelas is a much more serious disease when epidemic or endemic than when it occurs sporadically; and the "type" of the epidemic—as observed in other instances which have been watched to their termination—will be the basis for an opinion as to the probable issue of a particular case.

It is said that the wandering or "erratic" form of the disease is attended with considerable danger, but in my own experience cases have exhibited this character to a high degree and yet have not only terminated happily, but have never exhibited the slightest disposition to do otherwise.

The cases of Erysipelas which are the most dangerous are those which, commonly occurring after wounds of considerable extent—either accidentally or scientifically produced—are attended with much diffuse cellular inflammation; and which, finding their way, if not from their commencement, at least very early in their history, into the hands of the surgeon, will not be described in this place.

Treatment.—As I believe that the class of cases which have been described in such manner as to justify the use of antiphlogistic treatment, do not exist except in the histories of the past and the imaginations of the present, it appears to me unnecessary to say how much blood should be taken from the arm of a man, provided that he is found in a condition that we never meet with.

The *general* medical treatment of Erysipelas resembles rather that of the adynamic fevers than of inflammations, even supposing that the latter should present occasionally what is called a "sthenic" form. Almost all the cases—so far as my own experience reaches, all—that come under the care of the physician from the first, not only bear well, but are positively benefited by, supporting and tonic treatment. The kind and degree of such treatment must be determined by, and proportionate to, the severity of the symptoms which have been already described.

In some cases, stimulants are required from the first, the conditions which necessitate their employment being identical with those which are common to that large group of diseases in which Erysipelas finds its place. When stimulants are not required at the onset, little or nothing is gained by such use of salines, or any other general treatment, as shall do more than maintain a normal amount of the secretions. Thus, in very mild cases, in persons of average health, one or two doses of the simplest saline aperient may be all that is requisite. When the disease is more severe, and exhibits a tendency to spread after the balance of secretion has been restored, the patient at the same time becoming restless and exhausted, the most efficacious general treatment consists in the administration of bark with ammonia, during the day, and an efficient but not heroic opiate at night. Should the adynamic symptoms increase, large quantities of alcoholic stimulant are required at short intervals; and the amount that may be taken with advantage is as large as that which has been found useful in any of the specific fevers.

The tincture of the sesquichloride of iron of the London Pharmacopœia is by far the most useful medicine that I know of in the treatment of these cases. So marked is its action that it has been thought by some to exert a "specific" influence in Erysipelas; but without asserting that it possesses such power in the strict sense of the word "specific," it may be well to mention that its utility appears equally great in diphtheria, and perhaps still greater in cases of diphtheroid sore-throat. The essential condition of its success is its administration in large and quickly repeated doses; it has often happened that disappointment has arisen in the use of this tincture of iron, but in most of these instances the tincture has been given in doses of ten or fifteen minims three times daily, and such doses are certainly useless. But when the tincture is given in doses of forty minims, or even more, every four hours, the results have usually been most favourable. The most convenient form for its administration is a mixture containing in each dose forty minims of the tincture with an equal quantity of spirit of chloroform and glycerine, with an ounce and a half of water. The effects of this medicine may be seen sometimes after the first, often after the second, dose: the local inflammation ceases to extend; the inflamed part becomes paler, less tender, less swollen; the feeling of exhaustion is diminished, and with it such symptoms of exhaustion as exaggerated frequency of pulse, and dry brown tongue; the temperature falls; and sleep frequently ensues. As soon as such changes take place the quantity of the tincture may be reduced. It is not, however, safe to trust to this medicine alone; alcoholic stimulant is often required at the same time, but the action of the former has been too obvious in numerous cases for it to be confounded with that of the latter, or to be mistaken for those curative processes which occur in the natural history of the disease.

In the *local* treatment of Erysipelas two things are to be strenuously avoided; the one anything which shall expose the skin to variations of temperature, and the other anything which shall interrupt its natural function. Among the former are included exposure to draughts, and to the chilling effect of wet applications ; among the latter the covering of the skin with any oily matters, ointments, &c. It has occurred to me frequently to see Erysipelas spreading rapidly under the use of "cooling lotions," and to see it arrested by their discontinuance, and the application of simple dry flour, violet powder, or oxide of zinc—the inflamed part being sometimes covered lightly with dry cotton wool; the latter, however, being really necessary only when wishing to protect the patient from such draughts of air as are almost unavoidable in any large rooms, and which are sometimes quite unavoidable, and absolutely pernicious to certain classes of cases which find their way into the very well-ventilated wards of hospitals.

The application of collodion has appeared to me of use only when the Erysipelas has been closely limited in extent. Its application over a large surface has not only failed to do good, but, in consequence of its cracking and leaving rough edges, has done positive harm.

Various attempts have been made to arrest the spread of the inflammation, by some applications to the sound skin in the direction of, but beyond, its extending margin. The most approved of these has been, and is, the application of nitrate of silver, and there are facts to warrant this approval. It is desirable, however, not to attach too much importance to this measure. When nitrate of silver is applied in such manner as to affect the integrity of the true skin, I have seen Erysipelas start from the line of its application as from a new focus; and when, on the other hand, this evil has been carefully avoided, I have seen the Erysipelas extend through the line upon which it had been used.

As already stated, the tendency to death from Erysipelas exists in three principal directions. To that by asthenia I have already referred ; the other two which are most important, are either by implication of the brain, or by obstruction to the respiration. In the former the symptoms may be due to the blood-poisoning, or to extension of the inflammation to the meninges. When the cerebral symptoms are referable to the condition of the blood, no change in the treatment is required; but when to meningitis or meningeal congestion—supposing that this diagnosis can be accurately made—the application of ice to the head, and warmth to the extremities, together with free purgation, will be found useful. It is certain that such measures will relieve and lead to the cure of many cases of a most unpromising appearance.

When the danger to life depends upon interference with the respiration, either the fauces or the glottis is the most usual seat of mischief. It is then necessary to relieve, if possible, the swollen mucous membrane, and so allow of the passage of air through the larynx ; but when this cannot be accomplished, the surgeon should be ready to perform laryngotomy or tracheotomy.

When pus is known or is supposed to exist, even in small quantity, in the neighbourhood of important organs, such as the eye or the glottis, it is necessary that incisions should be made for its evacuation. When it exists in larger quantity under the skin of limbs, in the neighbourhood of joints, or glands, the same plan should be adopted; and *à fortiori*, when large tracts of cellular tissue are so affected, and the case resembles that of "diffuse cellular inflammation," free incisions are necessary. For all the details of the treatment of Erysipelas, when thus requiring surgical interference, the reader is referred to systematic and other treatises on surgery.

In conclusion, it need only be stated that all those hygienic measures, such as good feeding, fresh air, and quiet, which are essential in the treatment of all acute specific diseases, are required and under similar direction in the treatment of Erysipelas.

VARIETIES.—The following terms have found their way into ancient and modern books, and have been used to denote the several so-called varieties of Erysipelas:—

E. neonatorum.
E. complicatum, et simplex.
E. idiopathicum, et symptomaticum.
E. verum, et spurium.
E. phlegmonosum, vesiculare, bullosum, &c.
E. acutum.
E. erraticum.
E. nervosum, œdematodes, &c.

The above list shows that the principles of nomenclature have varied widely; names having been constructed on the fact of their representing such conditions as,—the age of the individual attacked; the presence or absence of complications; the supposed mode of origin; the fact of the disease being Erysipelas or something else; the degree of the development of one or more anatomical elements of the disease; the time of its duration; the mode of its distribution; and the nature of its complications.

It does not appear to me to be necessary to explain these words; their enumeration is sufficient to show their unscientific character and practical inutility, and to lead towards the hope that they may not be perpetuated.

PYÆMIA.

By John Syer Bristowe, M.D.

The present article will be limited to the consideration of that morbid state of the system to which the term "Pyæmia" has of late years been generally applied. This morbid state is closely related to phlebitis; with which disease it was until recently confounded, and on which it undoubtedly often supervenes. It is related to those diseased processes which Virchow has named "thrombosis" and "embolia;" for the secondary deposits which characterise Pyæmia immediately depend, in great measure if not solely, on the obstruction by solid material of the vessels leading to the spots in which these deposits occur. It is related to erysipelas, and such like "unhealthy" inflammations; for not only does it occur as a sequela of these affections, but their neighbourhood serves often to induce Pyæmia in patients who but for this neighbourhood would have escaped. Again, it is related to puerperal fever; or, to speak more precisely, the loosely applied term "puerperal fever" includes, with many other diseases, a large number of cases of puerperal Pyæmia. And lastly it is related to several more or less well-defined morbid conditions of the system (septicæmia) brought on by the entrance into the blood, through the veins or lymphatics, of various non-specific animal poisons. These related affections will all be fully discussed in their proper places; and will be noticed here so far only as the due elucidation of the subject before us renders necessary.

I. Etymology.

The word "Pyæmia," derived from the two Greek words πῦον and αἷμα, signifies literally pus in the blood. Its English synonyms are "purulent absorption" and "purulent infection." Every one of these terms implies a theory, viz.—that an essential feature in the disease to which it relates is the presence in the circulating fluids of the elements of pus. In this respect they are all objectionable; for while some authors accept this theory of the disease, others (whose opinions are equally well entitled to respect) reject it, and to them the name is necessarily the embodiment of error. The term "Pyæmia" is nevertheless a convenient one; it has come to signify to the practising medical man, quite apart from all theoretical considerations, a form of disease attended with certain definite symptoms and certain definite anatomical lesions; and even if some equally euphonious but less objectionable term were now to be proposed, it is more than doubtful whether the inconvenience of its substitution would be attended with any adequate compensatory advantage. In this qualified sense the term "Pyæmia" will be used in the following pages.

II. DEFINITION OF PYÆMIA.

Pyæmia is a disease, originating often in contagion, and attacking for the most part those who are suffering from the results of serious injuries attended with wounds, or who have undergone grave surgical operations, or who are labouring under acute suppurative inflammation involving bones, or in whom parturition has recently occurred. Its onset is usually sudden, and marked by the occurrence of a severe rigor followed by profuse perspiration. Rigors succeeded by perspirations for the most part recur; the pulse becomes feeble, rapid, variable, often intermittent, the respirations shallow and frequent, and cough (attended or not with expectoration) commonly shows itself; the tongue generally becomes dry and furred; the appetite fails; and nausea, vomiting, and diarrhœa not unfrequently supervene; the surface generally soon gets sallow or even distinctly jaundiced; the patient acquires very much the aspect of a person suffering from enteric fever—delirium at night often comes on; but he remains for the most part conscious, at least when roused; and soon becomes excessively feeble and prostrate. It happens often that, in the progress of the malady, inflammation, or even suppuration, occurs in some accessible part or parts, especially in or about the joints and in the muscular and cellular tissues. The duration of Pyæmia is generally from about four to ten days; but it now and then becomes chronic, and may then last for several weeks or even longer. Its result is almost invariably fatal. The chief lesions discoverable after death are patches of hæmorrhage, or of inflammatory consolidation, or abscesses, scattered in various proportions among the different organs and tissues of the body, but occurring far more often and far more numerously in the lungs than elsewhere. The part antecedently affected is generally found in a state of suppuration, and unhealthily inflamed or sloughy.

The most characteristic features of Pyæmia seem to be:—*first*, its supervention on certain special conditions of the system; *second*, the occurrence of rigors with perspirations; *third*, the presence of jaundice; *fourth*, the formation of external abscesses; *fifth*, the great prostration and early death; and *sixth*, the occurrence of certain characteristic lesions, easily to be recognised after death.

III. PATHOLOGY OF PYÆMIA.

1. *Morbid Anatomy.*

Discarding, in our detailed account of the morbid anatomy of Pyæmia, those abnormal conditions of organs and tissues, which may be now and then discovered after death from this disease, but have only a fortuitous connexion with it, we will limit ourselves to the consideration of those morbid changes only which form, so to speak, an integral part of Pyæmia.

(*a*) *Morbid Anatomy considered generally.*—The lesions which characterise Pyæmia consist in local congestions, extravasations of blood, inflammatory deposits, abscesses, and necroses. Simple congestion is a phenomenon which is apt to disappear after death, or to be modified and masked by mere post-mortem changes; it is frequently observed during life to accompany superficial pyæmic inflammations, it is frequently recognised after death in the vicinity of so-called "secondary deposits," and doubtless as a rule it precedes all the more important changes which attend this disease. Congestion is therefore a real and important link in the chain of pyæmic events, but its presence or absence *post mortem* cannot in all cases be taken as trustworthy evidence of its presence or absence during life. Extravasations of blood are of almost constant occurrence; sometimes they appear as petechial spots or vibices; sometimes as clots infiltrating the tissues of organs—abruptly marginated and resembling patches of pulmonary apoplexy; sometimes they form decolorized fibrinous masses, much like the fibrinous "blocks" observed under other conditions in the spleen, in the liver, and in the kidney. Inflammatory deposits are rarely if ever absent; and may be associated or mixed up with other morbid conditions, such as extravasations of blood, or may occur independently of them. In the former case they often surround the extravasations, and in connexion with serous surfaces form distinct false membranes. In the latter case they constitute, in the lung, patches of lobular hepatization; and produce in other organs nearly equally well-marked changes. The term "secondary abscess" has been largely employed to designate the localized morbid processes taking place in the course of Pyæmia. And in most cases of Pyæmia abscesses doubtless exist. Sometimes, especially in joints and certain other places, suppuration takes place so instantaneously that the formation of pus would almost seem to be the very first evidence in them of a departure from the condition of health. But more commonly the formation of an abscess is distinctly a later process, supervening on the extravasation of blood or on the effusion of lymph, and commencing either at the margins of the diseased patch, or at some central point in it, or involving the patch simultaneously in its whole extent. In some cases the cavities which pass for abscesses would seem rather to be the results of circumscribed gangrene than of true suppuration; for they yield a gangrenous odour, and contain a soft shreddy material, more or less adherent to the parietes, and infiltrated with a dirty-looking fœtid puriform fluid. A few words will comprise all that need be here said generally in regard to the microscopic appearances observed in the several morbid conditions above described. The elements of blood, more or less modified, may of course be discovered in abundance in the hæmorrhagic patches; fibrillated lymph, of the usual character, may be recognised in the inflammatory deposits, especially in those occurring on serous surfaces; and in those deposits occupying the parenchyma of organs granular matter, exudation corpuscles, and compound granule cells are generally abundant; true pus cells are by no means infrequent in pyæmic abscesses, but the puriform fluid is sometimes found to consist of the debris of tissue, oily particles, and disintegrating cell-forms only.

Although the description which has just been given is generally applicable to pyæmic lesions, in whatever part of the body they may occur; there are yet so many differences between different organs in regard to the relative frequency with which they become involved, in regard to the relative frequency with which the various forms of lesion above enumerated affect them, so many minor differences dependent probably on structural and other peculiarities in the organs themselves, that a special description of these lesions, as they occur in the more important organs at least, can scarcely be avoided.

(*b*) *Morbid Anatomy of Lungs.*—In the lungs the diseased patches are scattered irregularly, but are generally most abundant in the lower part. Their numbers vary considerably; sometimes they are exceedingly numerous, sometimes not more than two or three are present; and occasionally, while one lung is affected, the other lung is quite healthy. Most of them abut more or less extensively on the surface of the lung. The individual patches range generally in size between that of a filbert and that of a pea. Sometimes they are smaller, and not infrequently larger; but in the latter case the increase of size is generally produced by the coalescence of contiguous patches. The characters of these patches vary very considerably. Sometimes they are distinctly apoplectic; that is to say, they are reddish-black, void of air, firm, abruptly marginated, yield blood-stained serum on pressure, and differ in no respect, but that of size, from the pulmonary extravasations due to mitral-valve disease. More frequently, although still distinctly apoplectic, they have undergone changes; they have become more or less decolorized, the margin has assumed a pale buff colour, and the more central portions a rusty or brownish hue; at the same time some degree of softening has generally taken place. This softening often begins in, and may be limited to, the outer buff-coloured layer, which then forms an interrupted puriform interval between the bulk of the diseased patch and the surrounding healthy tissues; at other times this process commences internally, probably in connexion with the bronchial passage leading into the affected portion of lung; at yet other times a more general softening takes place, and the whole patch comes to form an abscess-like cavity. Sometimes, though much more rarely, the patches of lung disease are rather pneumonic than apoplectic, and then strictly resemble the patches of lobular pneumonia which supervene on laryngitis, diphtheria, and other diseases obstructing the larger air-passages. Under these circumstances the patches vary considerably in colour, according to the relative degrees in which congestion and inflammatory deposits may be present in them, but are granular and have the ordinary aspect of inflamed lung tissue. Pneumonia of this kind, however, is more often combined with other pyæmic changes in the lung, than it is an uncomplicated phenomenon; sometimes surrounding apoplectic and other patches, and it may be combining several of these into a common mass of consolidation; sometimes forming independent patches scattered indiscriminately among them. Abscesses or collections of puriform matter are common, but very far from universal; often they are formed, as has been described, by the breaking down of clots, still more often they result from the purulent infiltration of pneumonic patches, and in both such cases are found in combination with patches of hæmorrhage, or of inflammation, or of both. But there are some cases of Pyæmia where abscesses alone, sometimes surrounded by solid infiltrated parietes, sometimes by breaking-down tissue, are discovered. In these cases it would almost seem that the process is different from the beginning; that in some of them the formation of pus takes place coincidently with, if not prior to, whatever other inflammatory changes may be found associated with it; that in others the diseased and puriform patches are the simple result of necrosial disintegration or sloughing.

The lung tissue in which the diseased patches are imbedded may be, and often is, healthy; but very frequently more or less of it is congested or œdematous or even carnified. The bronchial tubes mostly present an excess of secretion, and those which are directly connected with the diseased tracts often contain pus, and sometimes the rusty tenacious fluid which characterises pneumonia.

The sub-pleural tissue, especially that investing the lung, is generally the seat of extravasations of blood; and appears therefore studded more or less thickly and more or less irregularly with petechial spots and vibices. The surface of the pleura may be smooth and healthy-looking; or it may be invested in its whole extent by a layer of recently effused lymph; and the cavity may be occupied in a greater or less degree by transparent, opaline, or even distinctly purulent fluid. But, perhaps more commonly, the lung is only partially covered with lymph—each lump of pulmonary disease, which abuts on the surface, forming a centre of inflammation and of a disc of inflammatory exudation which is thickest at the centre and becomes thinner and thinner as it recedes from that point, until at length it ceases. This lymph presents a reticulated surface, and differs in no degree, microscopically or otherwise, from that of ordinary pleuritis.

(*c*) *Morbid Anatomy of Heart.*—The surface of the heart, like that of the lung, is often studded with extravasations of blood; and these are generally most abundant about its basal portion. Similar extravasations may also be seen in the substance of the muscular parietes, and beneath the endocardium. In the walls of the heart, too, may not infrequently be discovered (generally in the midst of extravasated blood, or at all events within a zone of congestion) yellowish spots, from the size perhaps of a horse-bean downwards, which consist either of muscular tissue infiltrated with some inflammatory exudation, or of a cavity full of pus or puriform fluid, or of broken-down and disintegrated tissue. The muscular fibres in and around these spots will be found under the microscope to be more or less broken into fragments, devoid of transverse markings, and studded thickly with minute oily molecules. The diseased patches in the heart vary much in number; sometimes not more than one is present, sometimes they are almost innumerable. They vary too as regards their position; perhaps they are most common about the base of the ventricles, but no part is free from liability to them, and they are sometimes found in the musculi papillares. As in the case of the lungs, so here, the patches of disease act as centres of inflammation. When they reach the pericardial surface, they induce inflammation in that membrane, with exudation of lymph; and when they reach the endocardial surface, they may lead to important changes in the endocardium itself. In the former case the exudation is of the same nature as that which occurs in connexion with the pleura; in the latter case the endocardium itself is apt to become thick and granular, from interstitial inflammatory deposit, and its free surface to be studded with so-called "vegetations." Occasionally vegetations become deposited upon the valves. Excepting the various morbid conditions just described, the heart is generally found quite healthy.

(*d*) *Morbid Anatomy of Liver.*—Jaundice is a marked, though not an invariable symptom of Pyæmia; yet nothing has been detected, *post mortem*, in the condition of the liver to explain its occurrence. Frerichs asserts[1] that "the bile ducts are open, and usually pour out a little thin secretion," and that "the organ itself is in most cases anæmic and dry." The liver in these cases has, in fact, a healthy appearance except in so far as it happens to be the seat of special pyæmic changes. These changes consist in congestions, inflammatory exudations, and localized disintegrations or suppurations. They have no special seat. The earliest condition of disease, and one that is often alone seen, is the presence of congested patches of a port-wine hue. These vary in size and shape, have often a superficial area of two or three square inches, dip to a greater or less extent into the substance of the organ, and for the most part include irregular patches of unnatural pallor. Such patches often differ, so far as can be ascertained, in colour only from the surrounding healthy tissues. Sometimes studding these patches, sometimes occurring independently of them, spots may be seen of an opaque buff colour, in which the liver tissue is evidently infiltrated and softened, and it may be broken down into a puriform pulp. These spots have usually around them a halo of congestion, and their contents consist sometimes chiefly of pus-corpuscles, sometimes of disintegrated liver substance only. But besides these, larger abscesses are not infrequently met with—abscesses the size of a filbert, a chestnut, a hen's egg, or of still larger dimensions. These generally contain a greenish-coloured purulent fluid; and are sometimes, judging both from their odour and from their appearance, distinctly gangrenous.

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[1] Klinik der Leber-Krankheiten: Sydenham Soc. Transl. vol. i. p. 162.

(*e*) *Morbid Anatomy of Spleen.*—When the spleen is secondarily affected in Pyæmia, the morbid appearances which it presents are very much like those observed in cases of heart disease. They consist generally either of circumscribed extravasations of blood, or of fibrinous "blocks," which are both often of considerable size. The "apoplectic" clots tend to become decolorized at the surface and to break down variously into a puriform pulp; the fibrinous blocks are usually softer and more juicy than those of heart disease, and tend, like the clots, to liquefy. Distinct abscesses, too, of various sizes are often scattered throughout the organ.

(*f*) *Morbid Anatomy of Kidneys.*—The kidneys are frequently involved; and the morbid changes which occur in them are observed both more frequently and to a greater extent in the cortex than in the medulla. The medulla, however, by no means escapes. Sometimes these changes are limited to a single spot in one kidney, sometimes they affect both organs almost universally. But more commonly they are present in both and in some intermediate degree of severity. Occasionally no abscesses have formed, but almost the whole tissue of the organ is mapped out by tracts and bands of deep congestion, which alternate with and surround patches, of which the colour is unnaturally pale. More frequently distinct abscesses are present; these are generally small and tend to become clustered; and both the individual abscesses and the groups of abscesses assume a linear arrangement, perpendicular to the surface of the kidney. The abscesses contain a distinctly purulent fluid, have invariably a margin of intense congestion, and vary generally in size from that of a pea or horse-bean to an extreme degree of minuteness. Sometimes, however, they are so large as to contain an ounce or two of pus. They can almost always be seen on removing the capsule of the organ, and very often the removal of the capsule allows the contents of the more superficial ones to escape. The formation of pus in these abscesses seems to take place, originally at least, in the intertubular tissue; and very often in the early stages the Malpighian bodies and tubules in the affected spot are quite healthy.

(*g*) *Morbid Anatomy of other Abdominal Organs and of Peritoneum.*—Congestions and petechial extravasations are apt to occur, both in the gastro-intestinal mucous membrane, and in that of the genito-urinary apparatus. As regards the first-named mucous tract, it is an interesting fact that there are occasionally observed upon it, and more especially on that part of it which belongs to the cæcum and colon, patches of granular exudation. Occasionally, too, the intestinal submucous tissue becomes the seat of well-marked pyæmic deposits, which may lead to the destruction of the mucous surface over them, and the production of a sloughy ulcer, not unlike the ulcer of enteric fever or that which follows the opening of a boil.

As regards other organs connected with the abdomen little need be said. They are rarely affected secondarily, and even when they are thus affected they present few points of importance or interest either to the pathologist or practitioner. Of them all, the prostate and the testicle probably most often undergo suppuration.

The pyæmic affections of the peritoneum resemble those of the pleura and pericardium. Sub-serous extravasations of blood are common; and inflammatory changes occurring within viscera (especially the liver and the spleen) lead to inflammation in the serous surface external to them, which may remain limited in extent, or become general peritonitis. Very often an abscess forms between the surface of the diseased lump and whatever organ or part is in contact with it—the abscess being limited laterally by adhesions, which correspond accurately to the margin of the lump. The same thing, though on a much more minute scale, is of general occurrence in the case of the lungs and pleuræ.

(*h*) *Morbid Anatomy of Brain.*—The brain does not appear to be a very frequent seat of pyæmic changes. When present, however, they consist of congestions with extravasations, of circumscribed softenings, and of abscesses. The extravasations affect chiefly the surface of the organ, and though perhaps generally petechial, sometimes become sufficiently abundant to occupy an extensive tract of the subarachnoid tissue. The circumscribed softenings and abscesses occupy indifferently any part of the brain,—the grey matter, the white matter, the cerebrum, the cerebellum, the corpora striata, the optic thalami, the pons Varolii,—no part necessarily escapes. The former are yellowish, more or less congested, more or less softened, patches, such as are met with in cases of so-called "embolism" of the brain, but of smaller size, varying mostly from that of a horse-bean downwards; the latter are distinct abscesses containing a glairy greenish-yellow pus, and sometimes attain considerable dimensions. The number of foci of disease present at one time varies very considerably; sometimes not more than one or two are discovered, sometimes they are so numerous that scarcely any part of the brain, so large even as a chestnut, is found free from them. The softened patches contain, in addition to disintegrated nervous tissue, vast numbers of compound granule cells.

(*i*) *Morbid Anatomy of Organs of Sense.*—Of the organs of sense the eye only calls for special remark. This organ occasionally becomes the seat of suppurative inflammation, especially in cases of puerperal Pyæmia. In these cases the affection of the eye is characterised[1] "by redness of the conjunctiva, intolerance of light, and contracted pupil; rapidly followed by opacity of the cornea, and excessive chemosis." The eye ultimately sloughs, and its contents escape.

(*k*) *Morbid Anatomy of Bones and Joints.*—Suppuration sometimes takes place in connexion with bones. The affected bone or portion of bone then becomes rapidly denuded of periosteum; fœtid pus accumulates upon its surface, while at the same time probably pus infiltrates its cancellous texture, and rapid necrosis ensues. The joints are much more frequently affected than the bones. The synovial fringes become intensely congested, and the synovia increased in quantity, or replaced by pus or puriform fluid. The capsule of the affected joint becomes distended, and the parts external to it become more or less inflamed. When the fluid within the joint assumes a purulent character, which is by no means always the case, it often happens that the parts of the lining membrane which had been congested become pale, that destruction of cartilage takes place, that the joint, in fact, becomes disorganized. All joints, small as well as large, are liable to be affected.

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[1] Arnott, Medico-Chirurgical Transactions, vol. xv.

(*l*) *Morbid Anatomy of Cellular Tissue and Muscles.*—Again, the secondary effects of Pyæmia show themselves constantly among the muscles and in the cellular tissue of the body generally. Extravasations of blood here are exceedingly common, inflammatory congestions and exudations frequently occur, and abscesses (often of large size) form rapidly, and almost without warning. These morbid changes are often observed in the walls of the chest and belly, and in the neighbourhood especially of joints, for inflammation of which latter parts they are then very liable to be mistaken. Pyæmic abscesses have been met with in the tongue.

(*m*) *Morbid Anatomy of Skin.*—The skin necessarily partakes sooner or later in any morbid process which is going on immediately below it; and hence discolouration of skin is frequently observed over superficial pyæmic infiltrations and abscesses. When jaundice is present, the skin necessarily partakes in the general icteroid tinge. Apart from the above, the morbid conditions of the skin in Pyæmia are not very important. Petechiæ are not very common; sudamina are frequently present; and occasionally vesicular and pustular eruptions have been observed.

(*n*) *Relative Frequency with which Organs are affected.*—There is considerable difference in the relative frequency with which the various organs and tissues of the body become secondarily affected in Pyæmia. The lungs rarely escape, and not infrequently are the only parts in which morbid changes are observed. The viscera affected next in frequency to the lungs would seem to be the kidneys. After these the liver, spleen, and heart. Then perhaps the brain. Among organs less often affected may be enumerated the intestinal canal, the testis, the prostate, and the eye. The joints and the general cellular tissue of the body become of course very frequently the seat of secondary affections. And indeed, from the great extent of the one and the great number of the other, disease in these parts is without doubt constantly overlooked. The serous membranes, at least one or two of them, are rarely found uninflamed: this condition may occasionally depend on morbid processes originating in themselves, but in the great majority of cases, as has been already explained, is due to the extension of inflammation from some subjacent organ.

(*o*) *Morbid Condition of Blood.*—The chemistry of the blood in

Pyæmia has not, so far as we know, been investigated. We must content ourselves therefore with the discussion of its physical properties. With certain important exceptions, which will be presently fully considered, the blood in Pyæmia presents no important differences from the blood of health; it retains its natural colour, the blood discs and the white corpuscles preserve their due numerical relation to each other and to the mass of the blood, and so far as can be recognised their normal characters. Sometimes the blood appears to be unusually fluid, to present in the cavities of the heart and in the larger vessels only traces of coagulum. More commonly it coagulates in the usual way; and we find distinct clots in one or more or all of the cavities of the heart. Sometimes these are ordinary-coloured post-mortem clots, sometimes they consist wholly or almost wholly of pure fibrine; and in either case they may be prolonged in a cylindrical form into the large vessels. There is no special tendency for the right side of the heart to be occupied, far less to be occupied exclusively, by these clots. They may be found there, it is true, and found there while the left cavities are contracted and empty. But in many cases, while the right side is empty, the left is distended with them. In fact there is nothing in the situation or character of these clots to distinguish them from those which are found in many other forms of disease. The adherent rounded clots, which soften in their interior into a puriform pulp, have sometimes been supposed to characterise Pyæmia. But this is clearly a mistake.[1] Such clots are far more commonly observed in other cases; indeed are altogether exceptional in Pyæmia; and when present are evidently accidental and probably trivial complications.

The most important and characteristic changes of the blood in pyæmic cases are manifested more particularly in the veins of the part at which infection is supposed to have occurred, and in the small arterial twigs leading to the spots in which secondary lesions have become developed. The veins leading from the seat of supposed infection have been examined over and over again with extreme care; and the general results of these examinations may be shortly summarized. In some cases the veins, though traced into suppurating and even sloughing regions, are found, both as regards their walls and their contents, apparently entirely healthy; in other and more numerous cases they are seen to be in various ways and degrees diseased. Their parietes are thickened and indurated; they may be seen to communicate by orifices, resulting from ulceration or some other cause, with the morbific elements in which they are lying imbedded; and their interior is occupied by coagula. These coagula are mostly adherent, and more or less decolourized; they may be solid throughout, but more commonly are reduced in their interior into a reddish or yellowish puriform pulp or fluid. This fluid appears generally to consist merely of disintegrated fibrine, but in some cases is undoubtedly true pus.[1] It is mostly, but not always, separated from the venous walls by a layer of still consistent fibrine; and is generally shut out from the proximal portion of the venous channel in which it lies by a continuation of this layer of fibrine, which forms a kind of septum or diaphragm between them. Generally the rounded extremity of the hollow fibrinous cylinder thus formed has adherent to it and prolonged from it a process of ordinary coloured clot. Sometimes one, sometimes several veins are found thus affected, and sometimes a considerable length of one is converted into an elongated abscess.

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[1] See Papers on Softening Clots in the Heart, in the Transactions of the Pathological Society of London. Vol. vii. p. 134, and vol. xiv. p. 71.

The ultimate arterial twigs, distributed in the lungs to the masses of diseased lung structure, seem to be invariably occupied, indeed distended, by a soft pulpy yellowish material, or by something more nearly approaching to ordinary coagulum. This material is found to consist mostly of mere disintegrated fibrine presenting the debris only of cells. But sometimes it contains distinct pus[2]—that is to say cells resembling in all their visible characters pus-corpuscles or the white corpuscles of the blood, but so abundant and so closely aggregated as wholly to negative the notion of their being normal blood-elements. Similar coagula have been detected in the small vessels leading to the diseased patches occurring in other organs besides the lungs—in the vessels for example of the heart, the spleen, and the kidneys.

(*p*) *Connexion between blocked-up Condition of Vessels and Secondary Deposits.*—So constantly are these coagula found in the small arteries, if looked for carefully, that the conclusion is forced upon pathologists that there is between them and the patches of diseased tissue a relationship of cause and effect. It might be surmised that the coagula in the blood-vessels are secondary to the local pyæmic formations with which they are connected, due to obstruction in the capillaries of the affected part and consequent stagnation of blood in the vessels leading to them. The characters of the coagula show however that this cannot be the true explanation of their mode of formation. They are not mere coagulated blood, nor even mere coagulated fibrine; but, if fibrine at all, are fibrine which has undergone changes, requiring time for their production, and often in point of time clearly in advance of the changes which have taken place in the patches of diseased tissue. There can be no doubt indeed that the sequence of events occurs in the reverse order; that the small afferent vessels become blocked up, and that on this blocking up supervene those changes which, according to circumstances, end in extravasation of blood, inflammation, purulent infiltration, or gangrene. This view is partly based on direct observation in Pyæmia itself, partly on corroborative evidence derived from other sources.

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[1] The following case bears out the statement in the text. A man died of erysipelas of the face and Pyæmia. "The brawny tissue of the face was infiltrated with pus, and pus oozed from numerous divided vessels. The facial vein of the right side was thickened and surrounded by indurated adherent tissue, and its canal was dilated. The first inch of its course was occupied by thick purulent fluid; to this succeeded a cylindrical adherent fibrinous coagulum, the distal extremity of which formed a hollow cone. The lower or proximal extremity gradually dwindled away, and was succeeded by purulent fluid: this latter continued throughout the rest of the facial vein as far as its junction with the jugular. No coagulum or adhesion separated this fluid from the general circulation. The purulent fluid found in the veins presented under the microscope large numbers of corpuscles, but they were mostly smaller and more irregular in shape and size than normal pus corpuscles; and few, if any, presented division of their contents under the influence of acetic acid. There were numerous secondary abscesses in the lungs."—*Manuscript Notes of Post-Mortem Examinations, St. Thomas's Hospital,* Nov. 2, 1857.

[2] See Transactions of Path. Soc. of London, vol. xiii. pp. 203, 204. See also Dr. Wilks's Report on Pyæmia, in the Guy's Hospital Reports, vol. vii. 1861.

Thus, it has been clearly established by experiments that if a small artery leading to any spot be obstructed, that spot becomes the seat of congestion and inflammation. The careful experiments of Mr. Wharton Jones[1] show that if in the frog's foot such an artery be divided, and the capillary area to which it leads be thus cut off from all direct supply of blood, these capillaries nevertheless become filled with blood by regurgitation from neighbouring anastomosing vessels; and further, that since by the same operation they have been cut off from the direct influence of the heart's systole, the blood which is poured into them becomes stagnant there, and intense congestion results. Again, the experiments of Cruveilhier, Sedillot,[2] Henry Lee,[3] and others show that, if mercury, oil, pus, fibrine, be injected into the veins, they become impacted in the small arteries connected with the network of capillaries next beyond the seat of operation, occlude them, and induce congestion, if not hæmorrhage, and inflammation in the respective areas to which they lead. Each of these experiments has no doubt some point of special interest, but all concur in establishing one common fact of fundamental importance, viz. that the sudden stoppage of the direct supply of blood to a limited area tends to the production in that area of congestion and inflammation—of the very processes in fact which mark the secondary effects of Pyæmia.

The analogies afforded by other forms of disease are still more to the point. When arteries become obstructed either from morbid changes in their walls, or from plugs in their interior, the parts which they supply fall into an unhealthy condition. It is needless to dwell upon the changes which take place in the lower extremities, when in old age the arteries become closed by accumulated atheromatous and earthy deposit, or even upon the circumscribed softenings in the substance of the heart (leading to rupture), which attend similar changes in branches of the coronary artery. The effects however of thrombi and emboli must be considered a little more in detail. It has been clearly ascertained by the researches of Virchow, Kirkes, and succeeding observers that, in cases of heart disease with vegetations on the valves, these vegetations are apt to be detached, carried with the onward current of the blood, and impacted in the first artery they reach which is too small to permit of their transit. It has been clearly ascertained that such detached fragments, or "emboli" as they are termed, become fixed in the arteries of the brain (more especially in the middle cerebral artery), and lead, in the brain structure beyond, to circumscribed congestion, inflammatory softening, and disintegration of tissue; that they become fixed in the small arteries of the spleen, and lead to extravasations of blood and so-called "fibrinous blocks;" that they become fixed in the renal arteries, and lead at one time to exudation of blood and lymph, at another time to minute abscesses; that they become fixed in the arteries of the retina, and lead to similar results there; that in fact they may occlude any artery of any organ, and thus lead to specific changes in the bit of tissue which that artery supplies. It has been clearly ascertained also that clots or "thrombi" formed in the interior of veins break down and crumble; and that their fragments, swept away by the stream of the blood, pass onwards with it from the smaller to the larger veins, through the cardiac cavities and orifices, and thence still onwards along the arteries, until, like the broken off cardiac vegetations, they become impacted, and by their impaction produce identical results. Further, it has been ascertained that clots form spontaneously, so to speak, not in the veins alone, but in the heart's cavities, and in the arterial system; and that the clots thus formed in the latter situation occlude the arteries in which they arise, and lead in the parts beyond to the same changes as have been described in connexion with emboli. Here again, throughout the whole series of allied but not identical processes, we find that obstruction of the supplying artery causes in the part supplied precisely those lesions which occur as the specific local manifestations of Pyæmia.

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[1] Astley Cooper Prize Essay, "On the State of the Blood and Blood-vessels in Inflammation." Guy's Hospital Reports, Second Series, vol. vii. p. 23 et seq.

Sedillot, De l'Infection purulente.

[3] Lee, On Phlebitis.

It has been already stated that the secondary effects of thrombosis and embolia are identical with those of Pyæmia; and essentially no doubt they are so. Yet there are between them certain minor differences—differences chiefly of degree—which it may be desirable to consider. This will be most conveniently done by taking three or four important organs and comparing the effects of these diseases upon them. In the brain the influence of embolia or thrombosis is almost invariably limited to a single spot; Pyæmia produces many spots of disease. The region affected in embolia is generally larger, at least in the beginning, than the individual regions affected in Pyæmia. Moreover, in the former case breaking down of tissue is far less rapid than in the latter case, and actual suppuration rarely if ever occurs. The pulmonary apoplexy attendant on heart disease is we believe generally, if not always, due immediately to thrombosis of branches of the pulmonary artery belonging to the apoplectic region, followed by congestion and rupture of the capillary network of the part. Now the clots of ordinary pulmonary apoplexy are almost identical with the pulmonary clots of certain cases of Pyæmia. Like them they become decolourized upon the surface, like them they may become more or less perfectly surrounded by a rim of softening or suppuration, or may present similar changes in their interior, and like them, when they abut on the surface of the lung, they lead to the deposition of a layer of fibrine on the overlying pleural lamina. But as a rule they are less numerous, and individually much larger, than pyæmic clots; and their tendency to soften, to suppurate, to slough, is far less. In the spleen, the wedges or blocks of effused blood, or fibrinous exudation, which so commonly result from cardiac emboli, are in their general aspect almost exactly like those connected with Pyæmia; but here again the tendency to rapid suppuration or decomposition distinguishes for the most part the one form of deposit from the other. In the case of the kidneys the differences are less pronounced: in both affections minute abscesses are of common and early occurrence; in both, hæmorrhages and exudations of fibrine alone are occasionally met with. It would seem then that the chief distinction, between pyæmic deposits and those resulting from simple embolia, resides in the fact of the greater tendency of the former to undergo changes of degeneration and destruction. But this after all is chiefly a difference of degree; and the difference in this respect between them is no greater than the difference which may often be observed between actual cases of Pyæmia. Thus in one case of Pyæmia hæmorrhagic effusions only will be discovered in the internal organs, in another case patches of inflammation only, in a third abscesses, in a fourth gangrenous excavations; though more commonly doubtless these various conditions are to a greater or less extent commingled.

2. *General Pathology of Pyæmia.*

(*a*) *Conditions of System essential for the Development of Pyæmia.*—Exclusive of a few cases in which, from want of a trustworthy history or from some other cause, it has been impossible to determine what has been the original seat of disease; and of a few other cases which may be found recorded, wherein after very minute and careful investigation nothing that could be regarded as a starting-point for Pyæmia has been discovered, and which may possibly, therefore, have been idiopathic—exclusive of these, all cases of Pyæmia appear to take their origin in some one or more well-marked local conditions of disease; some coming naturally under the care of the physician, others under the care of the accoucheur, others under the care of the surgeon. We will enumerate them without particular reference to the department of practice to which they respectively belong. *First.*—Pyæmia frequently follows on accidental injuries, such as extensive burns or scalds, bruising and laceration of tissues, and compound fractures, especially on fractures of the long bones, and of the bones of the head and pelvis. Such accidents often, of course, become repaired without any untoward complication; often they are followed by serious results, and even death, quite independently of anything approaching to Pyæmia; but often, and even at a time when they appear to be progressing favourably, the symptoms of Pyæmia come on and the patient dies rapidly of this disease.

The occurrence of Pyæmia in these cases is generally distinctly preceded by sloughing, unhealthy suppuration, by erysipelatous inflammation, or some allied process. *Second.*—Pyæmia is the bane of certain operations. No operation possibly can be regarded as absolutely free from liability to the supervention of Pyæmia; but large operations, operations that is to say which leave extensive raw surfaces, especially therefore amputations of the larger limbs, operations too in which bones are involved, and operations in which certain parts (the bladder, prostate, and urethra, to wit) are implicated, are especially liable to the supervention of Pyæmia. But here again the pyæmic symptoms are mostly preceded by the occurrence of unhealthy processes at the seat of operation. *Third.*—Pyæmia is peculiarly apt to follow on acute suppuration taking place in connexion with bones. Sometimes from an accident (unattended by breach of surface), sometimes, so far as can be made out, spontaneously, acute inflammation is suddenly lit up in connexion with one of the bones—probably one of the long bones—suppuration rapidly takes place on the surface of the bone, between it and the periosteum, and in its interior; the bone dies; and in the course of these processes the symptoms of Pyæmia suddenly declare themselves. *Fourth.*—Phlebitis, as the disease is called, whether it be idiopathic or whether it be induced by injury or by operation,[1] is a pregnant cause of Pyæmia. This complication has been especially observed in connexion with the operation of phlebotomy, in operations on varicose veins and hæmorrhoids, and in connexion with the wounding or tying of large veins in the course of certain other operations. *Fifth.*—Pyæmia is by no means an uncommon sequela of suppuration involving certain of the organs of sense, such as the eye and the internal ear. *Sixth.*—So-called "low inflammations" attended with suppuration—in the male in connexion with the bladder, prostate, and urethra, and in the female in connexion with the ovaries and other genito-urinary organs—are not infrequently succeeded by Pyæmia. *Seventh.*—The period immediately after parturition is peculiarly obnoxious to the occurrence of Pyæmia. "Puerperal fever" is the generic term which is used for a variety of diseases occurring shortly after child-birth. What these diseases are it is not our province now to discuss; but certainly one form of so-called "puerperal fever" and one which causes no inconsiderable proportion of the deaths ascribed to puerperal fever, is Pyæmia. *Eighth.*—Certain forms of so-called "unhealthy" inflammation are not infrequently followed by Pyæmia. Such are phlegmonous erysipelas, diffuse cellular inflammation, carbuncle, dissecting wounds, malignant pustule. *Ninth.*—Pyæmia is described as taking place occasionally in the course of certain febrile affections, such as typhus, enteric fever, and variola.

Now, in reviewing the above series of cases in which specially Pyæmia is apt to occur, several facts come into prominent relief. It would seem in the first place that, in such cases, Pyæmia is almost invariably, if not always, preceded by some local suppuration, and that this suppuration is erysipelatous, gangrenous, or otherwise unhealthy. Such is the case after injuries, after operations, after affections of the bones, of the organs of sense, of the genito-urinary organs, of the veins: such too is certainly often the case in puerperal women. It would seem in the second place that Pyæmia is peculiarly apt to supervene in cases in which bones are involved in these morbid processes. It would seem further that in the great majority of cases, if not in all, there is reason to believe that veins are in some way or other specially implicated. Thus it has been conclusively determined that wounds and injuries of veins, and suppuration taking place in connexion with them, not infrequently lead to Pyæmia; it has been pointed out that the veins in the interior of bones are peculiarly thin-walled, and at the same time from their connexions prevented from readily contracting, and that when inflammatory processes are going on in the interior of bones these vessels are necessarily peculiarly implicated; as regards the skull, again, it is clear that the bones which form it, besides having in their interior the veins of the diploe, are related by their inner surface to the venous sinuses—channels which if not thin-walled like the veins of bones, are like them permanently patent; the eye and the ear stand in much the same position as the skull itself, they are bounded in fact by osseous tissue which almost necessarily becomes involved when serious inflammation occurs in the adjacent structures, and they communicate almost directly by special veins with the sinuses, the peculiarities of which have been pointed out; again the prostate and neighbouring parts are supplied with an almost superabundant net-work of veins; and lastly in parturient women the uterine portion of the placenta is provided with huge thin-walled venous sinuses which receive blood from the curling arteries and pour it into large uterine veins: at the time of parturition these are necessarily ruptured, and although by contraction of the uterus their orifices become in great measure closed, it is obvious that they are so circumstanced as to be peculiarly exposed to the influence of poisonous and other injurious processes going on in the interior of the uterus, or in connexion with its lining membrane.

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[1] Pyæmia is described as following on the operation of tying the funis in the new-born child.

(*b*) *Essential Cause of Pyæmia.*—We are now in a position to consider what is the essential cause of Pyæmia. The sudden onset of the disease, the markedly febrile and characteristic symptoms which it exhibits, the limited term of its duration, and its terrible mortality, together with the occurrence of specific lesions, all suggest a close analogy on the one hand between it and certain contagious fevers—typhus fever, enteric fever, small-pox, diphtheria, and the like; on the other hand, between it and certain diseases, such as glanders and hydrophobia, arising from the inoculation of animal poisons. In each of these analogous cases the disease is due to the entrance into the system of some morbid poison, to the circulation of this poison through the vessels with the blood, to chemical changes thereby induced in the blood, and through the agency of the blood in the system generally. In Pyæmia, too, it is manifest that the symptoms are due to the entrance into the blood of some *materies morbi,* and to changes thereby induced in that fluid and in the tissues through which it circulates. There are, however, marked points of difference as regards the mode in which the several classes of poisons above referred to enter the system. In contagious fevers it is mostly by the breath that the contagium takes effect; in glanders and in hydrophobia it is by inoculation; but Pyæmia, though in a certain sense contagious, is never imparted through the instrumentality of gaseous exhalations alone, and never even by inoculation, except the appropriate condition be present of a raw, suppurating, or sloughing surface. Further, though it may be imparted by some contagious influence, it may equally originate *de novo,* but never probably becomes developed even in the latter case except in connexion with some area of suppuration, and through the direct agency of that area. The poison which produces Pyæmia is evidently something more gross, something less subtle, than the poisons of those diseases with which we have compared it, and is capable only of acting on parts especially prepared as it were to receive and to develop it.

It is important to determine what this poison is, and how it gains an entrance into the system. It was at one time believed that pus, as such, is absorbed by the veins from the region of primary suppuration, and carried bodily to the various localities in which secondary accumulations of pus are discovered—that a true process of metastasis takes place. Again, it was imagined that this disease is simply phlebitis, in which the inflammation of the veins has extended to the vena cava and the heart. Arnott[1] maintained that the cause of what is now termed "Pyæmia" is "inflammation of the veins, the consequent production of pus in their cavities, and the entrance of this into the circulation." And since the period at which Mr. Arnott wrote, his views, with various more or less important modifications, have generally found acceptance. Thus, some have believed with Mr. Arnott that pus finds its way into the blood in consequence of the secretion of pus by the inner surface of some vein or veins inflamed by the extension of inflammation from surrounding parts; others have considered that pus is absorbed from some suppurating region by the open orifices of veins—orifices existing naturally (as in the uterus), or made by operation or disease; others, again, have supposed that the pus-corpuscles carried with the blood become arrested in the capillary vessels of the lung, and there produce the characteristic lesions of Pyæmia, either by multiplying by means of cell-growth and thus forming an abscess, or by acting as foreign bodies and thus inducing congestion and inflammation in the surrounding parts. In support of some at least of these views, it has been pointed out that the veins connected with the seat of primary disease are often thickened, and occupied by adherent coagula containing within them a puriform fluid; it has been maintained that pus-corpuscles may be recognised in the circulating fluid; and it has been found experimentally that the introduction of pus into the veins leads to changes in remote organs like those of Pyæmia. But Virchow[1] (whose researches in connexion with Pyæmia are most important) maintains that the puriform fluid in the affected veins is not pus, but simply disintegrated clot, that the clot is formed in the veins wholly independent of phlebitis, and that the diseased condition of the venous walls is not the cause, but (if related to it at all) the consequence, of the clot within it. Again, though Sedillot[2] has taken pains to show that pus-corpuscles circulate in the blood in cases of Pyæmia, and may by their microscopic characters be recognised there, it is now generally allowed that pus-corpuscles do not mingle with the blood in the manner supposed, and that even if they did it would be impossible to distinguish them from the white corpuscles of the blood itself. And lastly, although it has been shown that pus introduced artificially into the systemic veins may produce lobular inflammation of the lungs, it has been shown that the pus acts in such cases as an embolus, and much in the same way as other substances which lead to mechanical obstruction of the small pulmonary vessels. The theory which at present finds perhaps most general acceptance is that of which Virchow[3] is the chief exponent. He denies that in Pyæmia pus (meaning, by pus, pus-corpuscles) enters the blood; he denies that pus is ever found either in the thrombi occupying the veins of the region primarily diseased, or in the small vessels leading to the patches of secondary disease; he asserts that what had been regarded as pus is merely disintegrated fibrine, and that the material choking up the small afferent vessels of a secondarily-diseased tract is simply an embolus resulting from the crumbling away of the fibrinous material occupying the veins at the seat of primary disease; he maintains that all secondary pyæmic formations and changes are thus the result of embolia, but that the differences which these formations exhibit in different cases are due to the difference of process which has led to the disintegration of the original thrombus. So much with regard to his explanation of the mode of production of secondary pyæmic lesions: to explain, however, the general symptoms of Pyæmia, to explain certain diffused inflammatory processes (as inflammation of joints and of serous surfaces), which do not seem to be easily explicable on the embolic theory, he assumes that in many cases of Pyæmia, at least, certain ichorous juices are also absorbed into and act upon the system. Thus, according to Virchow, it would appear that Pyæmia is a complex condition; that from the veins at the seat of primary disease solid matters and poisonous fluids are circulated throughout the system; that the solid matters lead to the more material secondary lesions, the fluid matters to the more subtle changes, which combine to constitute the disease under consideration.

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[1] Med. Chir. Trans. vol. xv.

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[1] Virchow's Cellular Pathology, translated by Dr. Chance.

[2] De l'Infection purulente.

[3] Cellular Pathology, Lectures IX. and X.

These views are intelligible, and give a plausible explanation of most of the phenomena of Pyæmia; but they do not, we conceive, explain all the phenomena of the disease, neither, as it seems to us, are they based on an impartial appreciation of all the facts. We admit that in the majority of cases the puriform fluid in clots is simply disintegrated clot, but we maintain that the true pus is occasionally met with in venous clots and in the clots of arteries, and that pus is occasionally discovered *in transitu* in the blood—not, we allow, in the form of scattered pus-cells, but in that of soft pellets.[1] We see no sufficient reason to believe that veins do not share in the morbid changes which are going on around them, or that phlebitis may not exist at a time when no appreciable thickening of their walls has taken place;[2] indeed we have reason to believe that pus may be formed not only on the lining surface of these tubes, but even in the interior of clots adherent to them, by the communication to them of those tendencies to cell-production which are a part of the inflammatory process. We believe that in the above views undue importance has been attached to the embolic theory, too little to the independent formation of thrombi within the arterial system. We do not see how the embolic theory explains satisfactorily those cases of Pyæmia, starting from some portion of the systemic venous system, in which the lungs escape in great measure, or entirely, while secondary deposits are found, it may be, abundantly in other organs. Lastly, it seems to us as erroneous to regard the corpuscular element of pus only as pus, as it would be to attach that name to the liquor puris exclusively. Pus consists of both a solid and a fluid portion. On the whole, we are disposed to believe that, owing to some form of unhealthy process supervening in the region of primary disease, unhealthy pus or the element of unhealthy pus (call it ichor if you will) finds its way into the circulating fluid, and poisons it; that this poisoning partly shows itself in producing in the blood a tendency to coagulate in the smaller vessels, partly shows itself by inducing more subtle but even more serious effects upon the system at large. We are not disposed to deny that some of the local effects may really be due to embolism, some even to the impaction of coagulated masses of pus-cells; but we believe that thrombosis alone is the more general explanation of that obstruction of the minute vessels which leads to the secondary deposits.

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[1] The following case of malignant and rapidly fatal scarlet fever may be quoted in confirmation of this statement. "There were no pyæmic deposits; but the following was the condition of the blood in the heart's cavities. The left ventricle was empty, but all the other cavities were filled with largish fibrinous coagula. The greater part of the clot in the right ventricle consisted of perfectly decolourized, recently deposited fibrine, straw-coloured, elastic, and semi-transparent. Embedded in its substance were a few small opaque whitish masses, which looked like clots of older formation entangled in the substance of the more recent one. These increased in number towards the pulmonic valves, and were very numerous in the cylinder of clot occupying the trunk of the pulmonary artery and its left branch, rendering it in fact somewhat nodulated. All these masses had an opaque, buff-coloured, creamy aspect, were irregular in shape, and appeared in the majority of instances to consist of convoluted, folded, wrinkled, or twisted fragments, as though they had been formed and moulded in other parts, and had become entangled and compressed by the surrounding clot. The masses were somewhat soft, and could be separated readily from the fibrine investing them; and when separated some of them could be unfolded, but at the same time gave no clear indication as to what their original shape had been; some looked as though they might be collapsed bags, others were possibly cylindrical; none contained fluid. The same appearances were found in the right auricle. The left auricle contained, besides an ordinary clot, a single soft mass of the same kind as those that were found on the right side. Under the microscope the ordinary fibrinous coagula presented the usual characters of such formations; but the soft masses consisted entirely of corpuscles, which had the size and general characters of pus, and of which (under the action of acetic acid) the nuclei were divided into two or three spherules."—*Manuscript Notes of Post-Mortem Examinations, St. Thomas's Hospital*, September 15, 1858.

The soft opaque masses above described were very likely emboli carried from the seat of suppuration in the tonsil; but they were also, so at least it seems to the author, undoubtedly pus.

See also Transactions of the Path. Soc. of London, vol. ix. p. 279.

[2] If the presence of phlebitis is to be denied in all cases where there is an absence of thickening and congestion of the venous walls, it may with equal justice be denied that bronchitis has been present when the bronchial mucous membrane is found after death neither congested nor thickened. But in many cases of fatal bronchitis the mucous membrane itself looks quite or nearly healthy.

(*c*) *Cause of relative Frequency of Pyæmic Deposits in different Organs.*—On either view of the question, it is easy to understand why the lungs should be, as they generally are, first and most seriously affected. For since the majority of cases of Pyæmia originate in connexion with the systemic venous system, the poisonous matters which induce the disease must reach first the pulmonary capillaries; and in connexion with these, which act as a kind of filter and purifier, their effects are naturally earliest manifested. Further, since, as regards the circulation, the lungs may be regarded as the equivalent of the whole of the body besides, it is obvious that even if all the morbid effects of Pyæmia throughout the system were produced simultaneously, the lungs would be still (if the morbid processes in them held any relation to the amount of blood passing through them) the equivalent in quantity of pyæmic disease of all the rest of the body, and would therefore far surpass in their liability to secondary deposits any other one viscus. It is not so easy to understand the differences presented by other organs as regards their relative liability to disease; why, for example, the brain should so often escape, why the spleen, the liver, and the kidneys should so often suffer: all are equally exposed to the effect of emboli originating in the lung; all are equally liable, it might be supposed, to the formation of thrombi in their smaller vessels. To explain these differences there must, we imagine, be something in the character of the circulation, something in the formation of the various organs, which modify both the tendencies to morbid changes in the blood circulating in them, and the mode in which these morbid changes affect their tissues.

(*d*) *Cause of different Character of Pyæmic Deposits in different Cases.*—What, we may now ask, is the explanation of those differences as regards the character of the local deposits (described on a former page) which distinguish one case of Pyæmia from another, and cases of Pyæmia from cases of ordinary embolia and thrombosis? Something is doubtless due to the different dates at which patients die. In those persons who succumb early, local processes of disease have had but little opportunity to develop themselves; in those who die late the later stages of suppuration and sloughing have had ample time to become established. But this explanation does not apply to all cases; neither does it apply to the differences observed between cases of embolia and cases of Pyæmia. Here, as elsewhere, those subtle chemical changes (termed vital) so deeply interesting, so little understood, come into play. Why is it that when a cancerous growth has appeared in one part of the body, the whole system speedily becomes influenced, and diseased processes occurring elsewhere assume also the cancerous character? Why is it that when tubercular disease has manifested itself in one organ, the same form of disease ere long becomes developed in other organs? Why is it, again, that amongst all the varieties—shades—of cancer and of tubercle, that one variety which has first shown itself in any case is the pattern upon which the subsequent deposits of the same disease are formed? It would seem that morbid processes, limited in the beginning to one spot, influence the chemistry of the blood, and that of the system generally, and thus produce in the tissues a tendency to repeat, under the influence of exciting causes, those very morbid processes out of which the tendency arose. Again, certain conditions of unhealthiness, dependent on a variety of causes, give a type to the morbid changes accidentally occurring in different parts of the body. Thus, according to the former rule, the occurrence of gangrene at one part tends to the production of gangrene in other parts; the existence of suppuration in one corner of the system tends to render inflammation suppurative elsewhere: thus, according to the latter rule, a certain condition of system (as that accompanying typhus) is apt to favour the occurrence of gangrene, another condition of the system (as that accompanying convalescence from various febrile affections) to favour the formation of local collections of pus; and thus on one or other or both of these principles it doubtless depends that Pyæmia, which is mostly sequential on some localized mortification or suppuration of tissue, presents in the character of its secondary processes not only those differences which distinguish it from embolia and thrombosis, but those differences (not due to relative duration of disease) which distinguish cases of Pyæmia from each other.

(*e*) *Certain Varieties of Pyæmia considered.*—We may here add a few words in regard to certain real or supposed varieties of Pyæmia. Pyæmia is generally an acute and quickly fatal disease: and when this is its character, pyæmic deposits may almost invariably be found. The deposits however are not generally related numerically or otherwise to the severity of the case; and cases are sometimes observed which, judging from the symptoms during life and from other evidence, are truly cases of Pyæmia, yet in which no pyæmic deposits are discovered. It would seem that Pyæmia resembles the exanthematous fevers in this respect, viz. that the blood-poison occasionally produces death ere local lesions have had time to manifest themselves. Sometimes cases of Pyæmia become chronic; the evidence that such cases are pyæmic being furnished chiefly by the occurrence of successive suppurations, in joints,[1] in the cellular tissue, in the eye (it may be), and in other parts. Cases of this kind are sometimes observed after parturition, and such are some of the cases following on enteric and other fevers. The frequent occurrence of pyæmic deposits in the lungs only has suggested the possibility of a local Pyæmia—a Pyæmia in which the poisoned condition of the blood is confined within certain limits, and effects its secondary changes within those limits only. By such a local condition of Pyæmia, confined within the ramifications of the portal system, Dr. George Budd[2] has endeavoured to explain the occurrence of hepatic abscesses in cases of dysentery. This explanation of the frequent concurrence of dysentery and abscess of the liver is probably erroneous;[3] and, indeed, our knowledge of the progress of true Pyæmia does not justify us in admitting that the poison of Pyæmia can be limited in the manner suggested.

(*f*) *Origin of Pyæmia in Contagion.*—Pyæmia, which probably only occurs in the classes of cases which have been enumerated—cases presenting the common feature of some unhealthy suppurating surface—may arise in them either spontaneously or as the result of some contagious influence. Cases of spontaneous origin are not infrequent. Many of the cases in which (whether as the result of injury or not) acute suppuration rapidly involves some large portion of bone are cases in which there has been no previous ill-health, no exposure to morbific influences. Many of the cases occurring after compound fractures or after operations arise under personal and surrounding conditions of good health. Other cases are induced by modes of dressing wounds which prevent union by first intention and promote suppuration and unhealthy discharges. Many of the deaths ascribed to puerperal fever, and occurring sporadically, arise under similar conditions, and, so far as we can see, wholly independently of contagion. There are many cases however, and these are in all respects the most important, where the occurrence of Pyæmia is distinctly due to the agency of some contagium. These cases are particularly met with in surgical practice and in obstetrical practice. As regards surgical practice, it is well known that Pyæmia may be often absent from a hospital ward in which cases of serious accident and cases of operation are in course of treatment, and may continue absent for a considerable period; that after a while a case of Pyæmia, or a case of erysipelas, may be introduced into the ward or may originate within it, and that from that time operation case after operation case, accident case after accident case, may be attacked with pyæmic symptoms. There can be no doubt here that the spread of the disease is due to the presence in the ward of some contagious influence—not of a pyæmic contagium, for the disease cannot be excited in any patient who is not suffering from a wound, nor in any wound probably unless it have become first unhealthy; but of a contagium which excites first unhealthy processes in the wound, and, by means of these unhealthy processes, Pyæmia. This contagium originates not only in cases of Pyæmia, but also in cases of erysipelas, diffuse cellular inflammation, phagedænic processes, and the like, and in cadaveric poisons. There is no doubt that the accumulation of many wounds in a limited space not merely promotes the diffusion of such a poison, but serves even to engender it.[1] It is not improbable that the poison exerts an influence, to some extent at least, through atmospheric diffusion, and that this mode of spread is largely aided by overcrowding and bad ventilation. But Pyæmia arises even more frequently from actual inoculation of a healthy wound, either by the fingers of those who are engaged in attending on the sick, or by the dressings and appliances which are employed upon them. The same remarks, with scarcely any modification, apply to puerperal Pyæmia. Puerperal fever has been known over and over again to be conveyed by the clothes, and more particularly by the hands, of nurses and practitioners; and has been thus carried, not merely from other cases of puerperal fever, but from the poison of erysipelas and other unhealthy inflammations, and from the dead-house. Further, the fact of the heavy mortality from this disease in lying-in hospitals,[2] compared with the mortality from the same disease in patients treated at home, is well established; and the occasional terrible outbreaks of puerperal fever, which blacken the annals of all these institutions, bear witness, if not to its spontaneous origin therein, at all events to its virulence of contagion under circumstances favouring its spread.

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[1] Dr. Wilks, in the Guy's Hospital Reports, remarks that there is a special tendency in Pyæmia to produce inflammation of the joints; that in cases rapidly fatal this tendency has scarcely time to manifest itself; but that in chronic cases (cases, that is to say, in which the blood-poisoning has not been excessive and in which visceral inflammations have been but little pronounced) this special feature of Pyæmia has full time for its development.

[2] Budd, On Diseases of the Liver.

[3] See Frerichs, Clinical Treatise on Diseases of the Liver; Syd. Soc. Translation, vol. ii. p. 113 et seq.: also Trans. of Path. Soc. of London, vol. ix. p. 241.

(*g*) *Conditions modifying the Tendency to Pyæmia.*—It has been asserted that the presence of organic visceral diseases; the debility attending convalescence from various acute maladies; the cachexiæ which result from intemperate habits, from insufficient quantity and quality of food; untoward circumstances attendant occasionally on serious accidents and grave operations and parturition—shock, hæmorrhage, nervous depression, and the like; that these, and many other circumstances tending to impair the general health, predispose in various ways to the occurrence of Pyæmia. It is difficult either to prove or refute such assertions; it is difficult to believe that unhealthy conditions of system, however produced, should be without influence in favouring the attacks of Pyæmia and diseases related to it; but, on the other hand, it may be confidently asserted that the vast majority of pyæmic patients have not been suffering from chronic visceral diseases; that but few of those attacked are recovering from acute diseases; and that many, very many, victims of Pyæmia have enjoyed the best of health up to the moment of the accident or the operation or the disease which has exposed them to the danger of Pyæmia; and that even in many cases the wound (if wound there be) has been progressing favourably up to within a few days of the sudden onset of pyæmic symptoms. It may be added as regards the subjects of amputations, that many more in proportion die of Pyæmia of those whose limbs have been amputated for injuries than of those whose limbs have been amputated for disease—many more, therefore, in this particular case of those whose bodily health has been good until within a short time of the occurrence of Pyæmia, than of those who have been reduced by previously existing disease. Time of year, age, sex, have also been considered among predisposing causes. Season has probably no very important influence. No doubt adults more frequently suffer than children, and men than women; but whatever differences in these respects may be observed are certainly due in an overwhelming degree to the relative frequency of grave accidents in the respective sexes, and at the respective ages. Neither infants[1] nor the aged are exempt. Want of ventilation, and filth, are important predisposing causes; but in order to predispose it is necessary that the want of ventilation should co-exist with undue accumulation of traumatic or puerperal cases, or with the presence of unhealthy inflammatory processes; that the filth should comprise offensive and other animal discharges. Mere dirt, mere deficiency of ventilation, have not, so far as we know, a very obvious relationship with Pyæmia; at least this may be said with certainty in reference to the Pyæmia of puerperal women.

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[1] See on this subject Report on the Hospitals of the United Kingdom, by Dr. Bristowe and Mr. Holmes, in the 6th Report of the Med. Officer of the Privy Council.

[2] Consult again Dr. Bristowe's and Mr. Holmes's Report, and also Dr. Barnes's Lectures in the Lancet of February, 1865.

IV. SYMPTOMS OF PYÆMIA.

1. *Symptoms considered collectively.*

The symptoms which usher in an attack of Pyæmia are generally well marked, unless the condition of the patient, or the nature of the disease under which he is labouring at the time of its supervention, mask the pyæmic symptoms. In some cases the accidental injury, or the operation, or the puerperal process, seem to be going on quite satisfactorily, up to the very moment when Pyæmia manifests itself. In other cases the wound made by accident or operation has taken on for a shorter or longer period some unhealthy action —the discharge from it has become ichorous and offensive, the process of union has become arrested or has retrograded, or sloughing has attacked the part, and constitutional symptoms in sympathy with these local conditions have appeared; or, in the case of the puerperal female, the lochia have become scanty and offensive, or have ceased, the abdomen has perhaps become tender, and high febrile symptoms have shown themselves. But, whatever the previous condition of the patient may have been, whether it have been one of perfect health or not, the first symptom to attract attention is almost without exception a sudden, severe, and prolonged rigor, followed by profuse perspiration. The patient soon recovers from this, and may for a time appear so well that the fear inspired by the first rigor gives way to the hope that it has been a mere accidental phenomenon, of no serious import. But before long, it may be the next day or at some earlier period, the rigor returns with its after sweating stage; and again and again, at varying intervals, the rigors and sweats recur. In the course of a day or two the conjunctivæ and the skin assume a sallow tinge; the patient becomes dull and heavy, or it may be restless, and acquires very much the aspect and manner of a patient suffering from some form of continued fever. In company with the symptoms above described, or in succession to them, others of more or less importance show themselves. The pulse, which at the beginning may have been unchanged, becomes rapid, even excessively rapid, weak, and perhaps intermittent; and these evidences of feebleness of the pulse increase as the disease advances. The tongue is often clean at the outset, but soon becomes glazed and fissured or furred, and after a time dry and brown; the lips also become parched, and sordes accumulate probably about the teeth. The appetite disappears; the patient becomes thirsty; and often there is nausea or vomiting. Diarrhœa, attended with offensive stools, occurs very commonly. The respirations become shallow and frequent; cough often supervenes, attended it may be with pains in the chest, with evidences of consolidation or of excess of secretion into the bronchial tubes, and with expectoration. The skin, in the intervals between the rigors and perspirations, and after they have disappeared, is generally hot and dry, and may present sudamina, and even it is said a pustular eruption. The sallowness generally increases, and often before death amounts to well-marked jaundice. Pain and swellings in or around the joints, or in other parts of the cellular tissue, often present themselves, and pus may form in these situations rapidly. As the above symptoms develop themselves and the disease advances, the patient becomes excessively prostrate, his face becomes shrunk and generally pale, his mental functions become more and more disturbed and impaired, slight delirium comes on, and possibly coma, or, but very rarely, convulsions; and at the end of a short period, generally between four and ten days, he dies. During the progress of the pyæmic symptoms the primary seat of disease (even if it were apparently healthy up to the moment when Pyæmia supervened) assumes an unhealthy character. Sometimes Pyæmia takes a more chronic course; the symptoms are altogether less pronounced; the fever attending them resembles hectic fever, and abscesses form in the external parts, as the joints and the cellular tissue; and the patient sinks, perhaps after a few weeks, of exhaustion; or after a protracted convalescence, during which abscesses cease to form, is restored to health.

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[1] A child nine months old was recently admitted into St. Thomas's with Pyæmia following on acute necrosis.

2. *Symptoms considered in relation to the various Organs, &c.*

We will consider separately the symptoms referrible to different parts of the system.

(*a*) *Aspect, Skin, &c.*—The aspect of the pyæmic patient may vary a good deal; but for the most part it resembles that of one suffering from enteric fever, or typhus. At first it may be healthy-looking or nearly so, but soon it becomes heavy and oppressed. The face is sometimes highly flushed, sometimes extremely pallid, and these conditions often alternate. Towards the close of the disease, pallor generally becomes established; and the countenance, unless modified in its expression by delirium or other conditions, becomes shrunken and anxious, or settles down into the expressionless dull aspect of the last stages of febrile diseases. The rigors are some of the most marked and prominent symptoms of Pyæmia. Cases are sometimes observed in which they have either been slight and so have escaped notice, or in which they have been wholly wanting. But in the great majority of cases they cannot possibly be overlooked. They vary much in number and frequency. Sometimes they recur at short and irregular intervals; sometimes they are quotidian, and resemble, and have been mistaken for, attacks of ague; generally they cease after two or three days, and the subsequent progress of the case is free from them. Their duration varies; sometimes each shivering fit lasts for half-an-hour or so, sometimes for only a few minutes. They are always followed by profuse perspirations. The temperature of the body rises considerably during the rigors.[1] In the intervals, the skin is generally harsh and dry. Sudamina, as might be supposed, not infrequently appear and are sometimes surrounded by a zone of congestion. They may then by a careless observer be mistaken for the spots of typhus or of enteric fever. A pustular eruption has been described as of occasional occurrence, by both Mr. Henry Lee and Dr. Wilks.[2] Sometimes livid discolourations appear; but these correspond for the most part to subcutaneous abscesses, or to tracts of diffuse cellular inflammation; ecchymoses are rare on the surface of the body. In a large proportion of cases the skin and conjunctivæ become distinctly jaundiced. This is a very important and characteristic symptom. It generally comes on shortly after the first symptoms of Pyæmia have shown themselves, and continues to increase up to the fatal issue. The jaundice, however, rarely if ever becomes intense, and is often so slight that in a bad light or from hastiness of observation it may pass unnoticed.

(*b*) *Organs of Respiration.*—The respiratory movements early become, as in other febrile affections, hurried and shallow. And this condition generally becomes more pronounced as the disease advances. Then the respirations not infrequently amount to forty or fifty in the minute, and are sometimes more numerous than this. After a while they are apt to become moaning or groaning in character. It has been asserted that the odour of the breath is in these cases peculiar and characteristic. The respiratory acts assume the characters just described, independently of all pulmonary disease, and in cases where the lungs are not at all affected, or where the affection is so slight as not to have caused special symptoms. But in the greater number of pyæmic cases the lungs and pleuræ become secondarily affected; cough comes on, which may or may not be violent; secretion takes place from the bronchial mucous membrane, or fluids get poured out from the air-cells into the bronchial tubes; and the cough consequently becomes loose, and attended with expectoration, which may according to circumstances be simply mucous, or purulent, or even distinctly pneumonic. The local phenomena correspond more or less to the morbid processes taking place in the chest. Tracts of dulness may sometimes be recognised on percussion, and sometimes uniform dulness at the base; but partly from the scattered arrangement of the patches of pulmonary disease, and partly from the absence ordinarily of any large amount of effusion of fluid into the pleura, dulness is often scarcely or not at all recognisable. Pleural friction-sounds again may occasionally be detected; but owing probably to the limited extent of the false membranes, and to the shallowness of the breath movements, they are not heard so often as might be supposed. The local signs most commonly present are such sounds as are heard in bronchitis, viz. crepitation, often amounting to gurgling and rhonchus. Pleuritic stitches may be complained of.

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[1] John Simon, Holmes's System of Surgery, vol. i. p. 49.

[2] Op. cit.

(*c*) *Organs of Circulation.*—The pulse in Pyæmia is specially remarkable for its feebleness. At the onset of symptoms it may differ little in frequency or in any other respect from its previous healthy condition. But generally it becomes from the very beginning rapid, or if not rapid at least variable, so that the slightest exertion of mind or body raises it twenty or thirty or even forty beats in the minute. The rapidity of the pulse however is generally considerable, and its rapidity tends to increase as the disease advances; so that not infrequently the beats of the pulse amount to 140 or 160, and may even rise to upwards of 200, in the minute. With this increase of rapidity, and with this variableness, the pulse also becomes very small and very compressible; and very often as the patient's general debility increases the pulse becomes irregular and intermittent. There is nothing generally very characteristic in the cardiac phenomena. The action of the organ corresponds with that of the pulse; and the sounds, unless they become masked by other sounds, are healthy though feeble. Pericardial friction may be looked for, but will not always be heard even when pericarditis is present.

(*d*) *Organs of Digestion.*—The organs concerned in digestion always sympathise more or less with the general condition of the system.

The tongue in the beginning may be clean; but it soon assumes an unhealthy character. There is nothing uniform however as regards its condition. Sometimes it becomes morbidly red and glazed, and may be fissured; sometimes it becomes thickly furred; but generally its final condition is one of dryness and brownness. Nausea and vomiting are frequent but not invariable symptoms. They are often amongst the first to appear; but they may arise at any time in the progress of the case, and may persist throughout its whole duration. The appetite mostly fails early, and thirst is generally present. Sometimes however the patient retains his appetite for a day or two, and may be persuaded to take even a good deal of stimulus and nourishment throughout the whole course of his illness; and thirst is by no means necessarily excessive, nor is it always complained of. Diarrhœa often shows itself, and the stools are then described as being highly offensive. This complication belongs to no particular period of the disease, and may either be persistent or temporary. It is interesting to bear in mind, in connexion with this symptom, the tendency to slight inflammatory changes, and even to pyæmic deposits, manifested in cases of Pyæmia, by the intestinal mucous membrane. The frequent occurrence of jaundice has been already spoken of. That the sallow discolouration in these cases is jaundice is proved by its presence in the conjunctiva as well as the skin; and by the fact that bile-pigment has been recognised in the urine, in the serum of the blood, and in the effusions into serous cavities. The jaundice has no dependence on the formation of pyæmic deposits or abscesses in the liver, and is frequently present indeed when the liver seems to be altogether healthy. Frerichs[1] remarks that "to all appearances the jaundice is here the result of an impaired consumption of bile in the blood, arising from an abnormal condition of the metamorphic processes which go on in that fluid."

Abdominal pain is sometimes complained of; but generally it is local, and the result of inflammatory processes going on in the internal organs (as the liver and spleen) and of circumscribed peritonitis in connexion therewith. In puerperal Pyæmia, general peritonitis and tympanitis are more apt to occur than in other cases.

(*e*) *Genito-urinary Organs.*—There is little to say in regard to the genito-urinary organs. From the inflammatory processes which so often go on in the kidneys, it is not surprising that the urine is occasionally found albuminous. We might naturally expect to find occasionally in it blood or pus. Unless the uterus be the primary seat of disease, there are rarely, if ever, any important symptoms referrible to that organ.

(*f*) *Organs of Locomotion.*—It is a common thing in Pyæmia, especially in the more chronic cases, to have inflammation and suppuration occurring in joints, and in the cellular tissue, and in connexion with bones and other organs. The morbid anatomy of these processes has already been considered. It remains therefore only to add that these superficial abscesses are more common in the chronic than in the acute forms of the disease, that they often attain considerable dimensions, that their origin and progress are often attended with excruciating pain, and that it frequently happens that pain and swelling attack joints and other superficial parts, and subside without leading to any further mischief. Muscular debility is marked from the beginning, but generally soon becomes excessive.

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[1] Frerichs, op. cit. vol. i. 162.

(*g*) *Nervous System.*—The nervous symptoms which appear in connexion with Pyæmia are almost identical with those which accompany enteric fever or typhus. The patient is at first perhaps a little heavy and dull and drowsy; but generally he becomes ere long (more especially at night-time) restless and somewhat delirious; yet usually he can easily be recalled to his senses, and to this extent remains conscious up to the time of death. The cerebral symptoms vary however in different cases. Sometimes the patient has little or no delirium, and is perfectly rational throughout his illness. Sometimes the delirium becomes violent, and he may become partially or even wholly comatose before death. But generally when coma, and especially when convulsions or paralysis appear, there is some actual disease going on in the brain to account for these symptoms.

The evacuations may or may not be passed unconsciously.

3. *Further Considerations in regard to Pyæmia.*

(*a*) *Time at which Pyæmia arises, and Duration of Disease.*—As regards the time at which Pyæmia appears in relation to the state of system on which it supervenes, nothing very definite can be said. In cases of accident and operation Pyæmia may come on at any moment, from the time when a suppurating surface is first established until the wound is perfectly healed. In cases of carbuncle and of erysipelas Pyæmia probably does not supervene until suppuration has taken place. In cases of acute suppuration connected with bones, and acute necrosis, pyæmic symptoms are present sometimes almost from the first. In puerperal cases Pyæmia usually comes on between about the third or fourth and tenth or twelfth day after labour. The disease is generally very rapid in its course; occasionally its duration is limited to three or four days, more commonly it lasts from six to eight days, and it may be for a fortnight. In chronic cases, especially such as recover, the duration of the disease may be much protracted.

(*b*) *Prognosis and Mortality.*—The prognosis of Pyæmia is exceedingly unfavourable. In surgical practice nearly all pyæmic cases die; in midwifery practice a larger proportion probably recover. There is much difficulty however in arriving at the exact truth in reference to this point; for although the symptoms of a typical case of Pyæmia are collectively ample proof of the existence of this disease, there is no one symptom, like the rash of typhus or the exudation of diphtheria, absolutely distinctive, and no one symptom which is invariably present. Hence the diagnosis of the least well-marked cases of Pyæmia is not always to be relied upon; and as cases which are said to have recovered mostly belong to this class, there is generally some, more or less justifiable, room for doubt in regard to the true nature of cases which are recorded to have got well. Still, there can be no reasonable doubt that of cases of Pyæmia coming under the care of the surgeon a certain proportion recover. Now and then cases are met with, having most of the usual symptoms of Pyæmia, and in which it is at least reasonable to suppose that Pyæmia, in a mild form perhaps, exists, which yet escape from the toils in which they seem to be involved. The probability that such cases are truly pyæmic is enhanced by the fact that they are apt to occur in a ward in which Pyæmia is prevalent, and that in some of them abscesses in external parts appear from time to time during their progress, and stamp their real character. But cases of recovery, with or without the formation of external abscesses, are far more common in obstetrical cases; at least it is in lying-in hospitals chiefly that, during the epidemic prevalence of Pyæmia, recoveries not infrequently take place after the supervention of symptoms, which in other cases usher in a rapidly fatal illness. Nevertheless, the disease is one of the most fatal with which practitioners have to deal; its premonitory symptoms are ground for the gravest alarm, and from a fully-developed and unmistakeable attack recovery is almost quite hopeless.

(*c*) *Diagnosis.*—The diagnosis of Pyæmia is not generally difficult, if the circumstances of the case and the symptoms be all considered. Still, its own symptoms are often so mixed up with those of the disease out of which it arises, or with those of the complications which become developed during its progress, and are often so modified by them, that the Pyæmia may be recognised with difficulty or even wholly overlooked; and further, there are several diseases with the symptoms of which its own have a decided, and even close, affinity, and with which therefore it is apt to be confounded.

It would be impossible to enumerate, still more to discuss, the various conditions which mask the onset, and it may be the progress, of the disease. An example or two must suffice. A patient has had an injury to the skull; after a while rigors come on and perspirations; and, with these, cerebral symptoms. An abscess has probably formed beneath the skull. Now the symptoms here are almost, if not quite, identical with those of commencing Pyæmia. But whether Pyæmia has come on as well is a point that probably cannot be then determined. The further progress of the case may clear up the doubt, but not always. Again, a patient, suffering from a large carbuncle, or from extensive diffuse cellular inflammation, becomes pyæmic; but it is more than probable, if the patient be suffering largely at the time from "constitutional irritation," that the additional "constitutional irritation" due to Pyæmia will be inappreciable. So again in a case of acute deep-seated suppuration connected with some bone (say the femur), and so again in puerperal peritonitis, the symptoms of the primary disease may be so sudden and so severe, and at the same time in many respects so like those of Pyæmia, that the supervention of the latter disease is very apt to pass unobserved.

The diseases, which above all others Pyæmia resembles, are typhus and enteric fevers, internal acute inflammations (especially of the lungs), urethral and bladder affections in which the kidneys have become involved, and acute rheumatism. The resemblance to fever is proved by the fact that even surgeons of experience occasionally mistake Pyæmia for typhus or enteric fever. The general symptoms and the aspect of the pyæmic patient are indeed almost identical with the general symptoms and aspect belonging to the fevers just named, and the frequent presence of diarrhœa approximates Pyæmia particularly to enteric fever. The liability to error is necessarily much increased when the Pyæmia depends on some deep-seated suppuration, which possibly escapes detection. The differences however are generally well pronounced; the severe rigors and perspirations of Pyæmia have scarcely any counterpart in either form of fever, in which for the most part rigors are scarcely marked, and the skin is dry. Moreover, the eruptions characteristic of typhus and of enteric fever are absent in Pyæmia, and the jaundice which generally attends the latter disease is rarely present in either of the former. The morbid anatomy and the progress of the several diseases will suggest other marks of distinction. In pneumonia not only is the general aspect of the patient like that of a patient suffering from Pyæmia, but the rigors, the profuse perspirations, the jaundice, and even the diarrhœa, may all be present; while in Pyæmia more or less of the lung is mostly involved, and there may even be pneumonic expectoration. It is obvious, therefore, that there might be great difficulty, even impossibility, in distinguishing a case of pneumonia, secondary (say) to a compound fracture, from a case of Pyæmia, supervening on a similar injury. On the other hand, a case of Pyæmia, in which the source of pyæmic affection is not obvious, might without much carelessness be taken for a case of pneumonia. Again, when inflammation and suppuration of the kidney-structures come on as a result of vesical inflammation, or of any other disease obstructing the passage of urine, febrile disturbance with delirium follows, and the combined symptoms differ often but little from those of Pyæmia supervening on the same local diseases; and here the difficulty of distinguishing between them is often greatly enhanced by the fact that both forms of disease are not uncommon sequelæ of suppuration occurring about the neck and base of the bladder. Further, acute rheumatism has many features in common with Pyæmia: in both there are profuse perspirations; in both inflammation in connexion with bones and joints is common (and it must be recollected that joints often inflame in Pyæmia without suppurating, and that in some cases of Pyæmia pain in the course of a bone, with sub-periosteal suppuration, is the first evidence of disease); in both, again, pericardial complication is not infrequent. We have considered somewhat in detail the resemblances between the several diseases above enumerated and Pyæmia, partly because they are really striking, partly because we have known them lead to errors of diagnosis. We have not however dwelt generally on the points which serve to distinguish them, for to discuss these completely or even usefully here would be to forestall needlessly descriptions of diseases which will be fully given elsewhere. There are yet other affections which, under certain circumstances, Pyæmia may simulate: such are delirium tremens, tubercular meningitis, and other inflammatory conditions of the brain or its membranes, ague, &c. It is needless, however, to do more than mention them.

V. Treatment of Pyæmia.

The treatment of Pyæmia is exceedingly unsatisfactory. But, as in so many other instances, although the medical treatment of a case of the disease may be of little avail either to arrest or modify its course, preventive measures are often in the highest degree useful both against its origin and its spread.

1. *Prophylactic Treatment.*[1]

In considering the subject of preventive measures, the simplest plan will probably be to take the case of a private patient, on whom some grave operation—amputation, for example—has been performed. To take precautions against Pyæmia is to take precautions also against those other unhealthy conditions out of which Pyæmia mostly arises. To prevent, so far as may be, their occurrence it is important to maintain both the general health of the patient and the healthy progress of his wound. To this end the patient's strength should be supported by appropriate and adequate nourishment; pain, sleeplessness, and irritability should be treated with opiates and seclusion from needless visitors and intruders; ample ventilation should be secured, and in aid of this curtains and all unnecessary hangings should be removed; further, perfect cleanliness should be maintained, and especially all evacuations, all offensive discharges, all dressings, should be removed at the earliest opportunity, and never allowed to accumulate in the patient's room. As regards his wound, that should be lightly dressed and kept cool, and never treated with the abundant dressings which are employed in some foreign hospitals, and which there promote suppuration and, in our belief, erysipelas and Pyæmia. The dressings should be of the simplest kind, and neither these nor sponges and such like things which have been used for the purpose of cleansing the wound should be used a second time. If erysipelas or sloughing or suppuration ensues, the treatment appropriate to these conditions must be employed; in the case of sloughing, charcoal, carbolic acid, and other antiseptics are valuable applications; but above all things it is essential to allow early and very free escape of pus and ichorous fluids. The same remarks apply to the treatment which should be adopted in the case of compound fractures and other injuries.[1] And in regard to cases of deep-seated suppurations, acute necrosis, carbuncle, and diffuse cellular inflammation, there is no doubt that free and early incisions are especially important in preventing the supervention of Pyæmia. In hospitals, or other places where many sick are accumulated, the precautionary measures above insisted on become doubly important, especially those of ventilation and cleanliness. In discussing these two measures we open up the old subject of hospital construction and hygiene—a subject which, even if in many respects appropriate to the present article, is far too extensive to be considered even briefly here. It may however be stated generally, that there should be abundant cubical space to each bed, abundant space between the beds, plenty of ventilation by means of open windows, aided by open fireplaces; that the walls, the floors, the ceilings, should be kept scrupulously clean; that the wards should be periodically emptied; that water-closets should be so arranged as in no degree to infect the ward; that sponges should never be used in the cleansing of wounds—nor anything absorbent in fact which might be used on a second occasion or in the treatment of another case. But the danger of Pyæmia, incidental to wards or places in which many sick are accumulated, is less due to mere accumulation of sick than to undue accumulation of such as are suffering from certain forms of sickness, such namely as are suffering from open wounds, whether arising from accident or disease or design. The presence of what has been termed a "traumatic atmosphere" would seem to have quite a special influence over the development of Pyæmia, and for that reason the creation of a traumatic atmosphere should be as much as possible avoided. This may be effected partly by the means just adverted to—viz. free ventilation, and avoidance of overcrowding—but especially by so distributing traumatic cases as to allow of no undue accumulation of them in any one ward of a hospital, or generally in any one spot.

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[1] See Bristowe and Holmes, loc. cit.

The presence of erysipelas, diffuse cellular inflammation, phagedæna, or any other unhealthy inflammation, and especially the presence or recent occurrence of Pyæmia in a ward, should be the signal for redoubled vigilance in securing that all sanitary regulations are as far as possible systematically carried out. Then, especially, all danger of inoculating the healthy with the unhealthy secretions of the diseased should be most watchfully guarded against; all dressings, &c. should be destroyed the moment they are done with; and neither nurses, nor dressers, nor surgeons, should pass from attendance on those whose wounds are unhealthy, above all from handling their wounds, to attendance on those whose wounds are healthy, until by proper precautions their persons, and especially their hands, are thoroughly disinfected. It may be added further that no one should at any time come direct from the dissecting room or post-mortem theatre (especially if he have taken part in the dissections going on there) to the treatment of surgical cases, without thorough purification and disinfection. Again, when diseases of the kind above specified have been received into a ward, and especially if they have shown any disposition to spread, it may become necessary either to remove them thence, or, still better, to avoid receiving into that ward for a time all accident cases attended with wounds, and to avoid operations on patients who happen to be therein. But notwithstanding all such precautions, these diseases will sometimes be found to cling as it were to a ward. Under such circumstances it becomes absolutely necessary to empty and dismantle the ward, to purify it thoroughly, and to keep it unoccupied for a shorter or longer period. Sometimes those forms of disease out of which Pyæmia is apt to arise, sometimes Pyæmia itself, may (dependent apparently on atmospheric conditions, or at least on local conditions of insalubrity) prevail over a district. If such be the case, the importance of temporarily suspending the performance of operations in that district becomes obvious.

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[1] See Professor Lister's papers on the treatment of compound fractures, &c.

The various precautions, of which the importance in relation to surgical practice has just been discussed, are if possible of still greater importance in relation to the practice of midwifery. In sporadic midwifery (if the term may be thus used), as in sporadic surgery, precautions are less absolutely needed than where midwifery is concentrated, as it is in a lying-in hospital. But in the former case puerperal Pyæmia has been known over and over again to be conveyed by the nurse or the medical attendant, who have brought infection in their person or in their clothes either from other puerperal cases, or from cases of unhealthy inflammation, or from the dead-house. In the latter case (the case of lying-in hospitals) the presence, so to speak, of a "parturient atmosphere" intensifies the liability to Pyæmia as it does to other infectious puerperal diseases, and leads often to terrible mortality. There is no doubt that no attendant is justified in running the risk of conveying such infection from one patient to another, and that if he have from circumstances become a possible source of danger, he is bound for a while to abstain from midwifery practice. As regards lying-in hospitals, every possible precaution ought to be systematically taken; and on the very first appearance of infection they ought at once to be emptied and purified. But we are very strongly of opinion that such institutions are dangerous institutions, and ought not, unless under exceptional circumstances, to exist; certainly they are not required as schools of midwifery, certainly every poor woman, who has a home however mean in which to be delivered, will be far safer in that home than in a hospital.

It must not be forgotten, however, that Pyæmia is not limited to hospital practice; that even in hospitals it may arise quite independently of hospital influences; and that many cases originate in private quite independently, so far as we know, of external deleterious agencies. Against such cases prophylactic measures are of course out of the question.

2. *Medical Treatment.*

The treatment of a case of Pyæmia resolves itself into the treatment of the original lesion, the treatment of the disease, the treatment of the complications of the disease, the general hygienic treatment of the patient.

(*a*) *Treatment of Primary Lesion.*—As regards the original lesion, it has been shown that in cases where Pyæmia supervenes on wounds (whether from accident or from operation) the wounds have generally already assumed an unhealthy aspect. According to the nature of this unhealthy process must be the local treatment; but especially it would seem important that the parts should be kept clean and cool, that disinfectant applications should be employed, that free incisions to admit of the escape of pent-up pus should be made. In cases where the wound appears to be healthy, there is probably some deep-seated suppuration in progress; and here, though the exact seat of suppuration may be difficult to detect, it is most important that it should be detected, and the pus therein thoroughly evacuated. The same rule applies with equal force to those cases where the primary disease is an abscess involving some deep-seated bone, or tract of cellular tissue. The reasons, on which the adoption of the above plan of treatment is based, are *first*, that by this plan the further entrance of poisonous matters into the blood may possibly be obviated; *second* (and most important), that by its early adoption the entrance of poisonous matters may be wholly averted and a threatened attack of Pyæmia warded off. With the same view it was proposed to cauterize the superficial veins on the proximal side of the diseased part. As regards obstetrical cases, it has been asserted by some that puerperal fever is mostly preceded by an imperfectly contracted condition of the uterus; but both by those who hold this view, and by those who do not, the importance of securing complete contraction of the uterine fibres and consequent closure of the ruptured uterine veins, by the use of ergot, and other means, has been strongly urged. It has been recommended also to cleanse the cavity of the uterus by the injection of disinfectant and astringent fluids.

(*b*) *Curative Treatment.*—Of the curative treatment of Pyæmia we fear little is known. Various plans of treatment have been from time to time adopted, and all probably have by some been supposed to be beneficial, all have by the majority of practitioners been found useless. Some of these plans have been based on the notion of the elimination of the disease; others on the notion of introducing into the system substances capable of battling with the pyæmic poison in the blood itself, and overcoming it there; others have been based on analogy; and others have been wholly empirical. Thus, acting on the assumption that the perspirations and the diarrhœa of Pyæmia are efforts of nature to eliminate some morbid poison from the system, warm baths and diaphoretics have been employed by some; by others the diarrhœa has not only not been restrained, but has been encouraged by laxative and purgative medicines. Thus, too, from the resemblance which the remittent rigors and perspirations of Pyæmia sometimes bear to the more regular attacks of the same kind which characterise ague, it has been imagined that quinine and arsenic, which are certainly remedial in the case of ague, might be remedial in the case of Pyæmia. Thus, again, guided we presume by the acknowledged fact that ample ventilation is one of the most important preventives against Pyæmia, some have looked on fresh air as absolutely curative, and have exposed their pyæmic patients to all the winds of heaven. And thus, others regarding the disease as one of putridity, have treated it with various forms of antiseptic agents. It would be useless to argue seriatim against the above and other modes of treatment; it would be unwise to oppose *à priori* the trial of any as yet untried modes of treatment; suffice it to say that, so far as we know, the mortality of Pyæmia is just as high now as it ever has been, and the antidote to it remains to be discovered.

There is, however, one mode of treatment, suggested within the last few years by Professor Polli,[1] of Milan, which, from the scientific character of the investigations which led to its proposal, and the manner in which the proposal has been brought under the notice of the medical profession, deserves a respectful mention. It will be necessary to go a little into the history of Professor Polli's investigations, and to trace shortly the steps which led him to his final conclusions. He assumed with most other physicians that septic poisons introduced into the blood produce their injurious effects through acting on the blood as a kind of ferment; and he assumed, as again others have assumed, that if any substance could be introduced into the blood, which while not acting injuriously either on that fluid or on the system generally, would arrest this process of fermentation, the exhibition of such substance in cases of septic poisoning would not improbably be curative.

Having long studied the effects of sulphurous acid, he had ascertained that it is not merely a powerful antiseptic, but that it equally prevents the vinous fermentation, and those other fermentations by which starch is converted into glucose, by which the pancreatic juice acts on fatty substances, and emulsine on amygdaline; and he came to the conclusion that in sulphurous acid we possess a substance capable of arresting every form of catalytic action.

But sulphurous acid cannot with impunity be introduced into the animal economy. Hence Professor Polli sought for other agents which, while having the virtues of sulphurous acid, should be free from its disadvantages; and he found such agents in the compounds of sulphurous acid with soda, potash, magnesia, and lime. He found that these sulphites, equally with sulphurous acid, prevent all forms of fermentation; he found that they may be given safely in large doses over a considerable period of time; and he further found that when taken into the stomach they become absorbed, diffused throughout the system, and eliminated without undergoing any chemical change, or at most only a very partial chemical change.

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[1] See Dublin Journal of Medical Science, vol. xxxiii. p. 367, and vol. xxxvi. p. 470.

He assumed therefore that that power of preventing putrefaction and fermentation which they exercise outside the body they would exercise probably equally well within the body. And further experiments made by him upon the lower animals, by the introduction of putrid matters into their blood, and by putting them at the same time under the influence of the sulphites, led him to believe that he had in these agents discovered valuable remedies for the various forms of septicæmia. It would seem that large doses of these sulphites (from thirty to sixty grains three or four times daily) may be given with impunity. We are not aware whether this plan of treatment has been so largely tested as the promises it seems to hold out might justify. We know of one case of supposed Pyæmia in which it was believed to have wrought a cure, but we know that in the practice of Mr. Simon, at St. Thomas's Hospital, it has entirely failed.

(*c*) *Treatment of Symptoms and Complications.*—When a case of Pyæmia is under treatment, it always becomes a question of treating, in addition to the general disease, certain symptoms as they arise, and, it may be, certain of the secondary lesions. On this head, again, we fear there is little satisfactory to be said. Some have recommended, on theoretical grounds, that diarrhœa should be encouraged. There seem no valid grounds for this course; and certainly if the diarrhœa became excessive, we should recommend that it be restrained, either by remedies administered by the mouth or by opiate enemata or suppositories. Again the pulmonary symptoms may become sometimes exceedingly distressing; and then, although probably we have no means of either checking or curing the morbid processes going on in the lungs, we may by opiates or other sedative medicine, judiciously administered, render the symptoms more endurable; or it may be that expectorants, especially ammonia, may be of benefit. Of course if abscesses form in superficial parts, they should be early punctured. As regards other symptoms, and other complications, we have really nothing to say. Many of them will need no special treatment at all; and generally where special treatment seems to be required, the medical man must be guided by his general knowledge of his profession, and treat them as he would treat such complications arising in the course of fevers and other allied disorders; bearing in mind, however, that where he cannot cure, it is better as a rule to aim at soothing and quieting, than (in the hope of achieving some insignificant advantage over the outposts, so to speak, of his patient's disease) to adopt a fidgeting line of treatment, and so render his few remaining hours miserable.

(*d*) *Hygienic and Dietetic Treatment.*—We come lastly to the general management of pyæmic cases, that is to say, their management as regards diet, stimulus, and hygienic observances. It need scarcely perhaps be pointed out that observance of cleanliness and ventilation, which we insisted on as an important prophylactic measure, should be equally persisted in during the whole course of treatment of a case of

Pyæmia; that, further, the patient should never be oppressed unnecessarily with accumulation of bed-clothes—that he should, in fact, be kept cool—and that his comforts should be carefully considered. Pyæmic patients, as has been shown, become at an early period excessively feeble; and in most cases excessive prostration is the most prominent among the symptoms which usher in death. No doubt this debility is functional rather than the direct result of the waste and degeneration of tissue; the consequence and the indication of blood-poisoning, rather than of the want of either stimulus or food; and theoretically, therefore, is to be counteracted by antidotal treatment rather than by nutriment. Whether we possess any mode of treatment that can be regarded as really antidotal we have shown to be in the highest degree problematical; but we have in these cases excessive prostration to deal with, and we must deal with it as best we can. To this end, it is manifestly our duty to administer both food and stimulus, and to administer them as largely as the condition of the patient will admit. The patient's appetite is generally quite annulled; and often loathing of food and vomiting are present. These conditions render it, of course, often exceedingly difficult, and sometimes impossible, to carry out the objects we have in view; and they show the importance of selecting for administration those articles which are least liable to offend the stomach, and of administering these in small and if possible frequently repeated doses, rather than rarely and in large quantities at a time. It is not easy to lay down any rule with regard either to the nature or the amount of food and stimulus to be given. These points must be determined in each case according to its requirements. But it is important to give whatever is to be given systematically. As regards food, that which is in the form of fluid is generally most suitable, such as animal broths, eggs beaten up, milk, gruel, arrowroot, and the like. As regards stimulus, perhaps, considering the irritability of the stomach, brandy, sherry, madeira, diluted according to the patient's taste, are the most generally serviceable. But lighter wines will often be found grateful. We protest against that excessive exhibition (that "pouring in" as it is appropriately termed) of stimulus which it has lately been the fashion to practise.

We need perhaps scarcely add that when pyæmic cases become protracted, and especially when they show signs of convalescence, and during the progress of convalescence, dietetic treatment becomes of paramount importance, and tonics form important aids to that treatment.

MALARIAL FEVERS.

By W. C. Maclean, M.D.

Before entering on the description of the remarkable fevers which are to form the subject of the following article, it is necessary to premise a few observations on the peculiar poison which produces them. This poison, which gives a distinctive name to fevers with periodical returns, is everywhere recognised by the term Malaria. "When a climate is called unhealthy, in many cases it is simply meant that it is malarious." (Parkes.)

In this article it is intended only briefly to summarize the few facts relating to this poison which have been tolerably well ascertained.

No chemist has yet been able to demonstrate the existence of malaria. We assume its existence from certain observed effects on the organism, just as we do in the case of other poisons which produce certain specific diseases. Malaria is believed to be the product of organic decomposition in soils, whatever may happen to be their mineral composition; water is indispensable to the process, and a high temperature, although not absolutely necessary, greatly aids it.

It is generated in greatest abundance in marshes, which contain a high percentage of organic matter; hence the name by which it is familiarly known, viz. *marsh miasm.*

It is often found in sandy soils and arid-looking plains devoid of vegetation; but in all such cases the soil will be found to contain a considerable proportion of organic matter, and water will be found not far from the surface, either in the shape of subterraneous streams, or detained by a bed of clay below the sand, preventing its free passage and keeping up evaporation.

Malaria is also generated in hard rocks, such as granite and trap, in a disintegrating state. A notable example is the island of Hong Kong, which consists entirely of weathered and decaying granite. In such soils, so long as they are undisturbed, the existence of malaria may not be suspected. In the case of Hong Kong, for example, it was not until extensive excavations were made into the disintegrating granite for building purposes, that violent and fatal remittent fevers appeared.

Dr. Parkes mentions that the soil of Hong Kong contains less than two per cent. of organic matter, but quotes Friedel to the effect, that disintegrated granite, which is highly absorbent of water, becomes often permeated by a fungus, and suggests the possible relation between the development of this fungus and the production of malaria.

The air of marshes known for ages as malarious has been examined by chemists. Watery vapour and carbonic acid are always found in excess; and, under certain conditions, sulphuretted hydrogen. "Carburetted hydrogen is often present, and occasionally free hydrogen and ammonia, and, it is said, phosphoretted hydrogen." (Parkes.) Besides the above, "various vegetable matters and animals, floating in the air, are arrested when the air of marshes is drawn through water, or sulphuric acid, and débris of plants, infusoria, insects, and even, it is said, small crustaceæ, are found." (Parkes.)

Malaria acts with the greatest intensity on the human system in situations which are low and moist, abounding in vegetation undergoing decomposition, *e.g.* in jungly districts during or immediately after the rainy season, at the bases of great mountain ranges, and in those belts of country in India termed *terrais,* formed by the débris of mountains rich in organic matter, which retain a large quantity of water and are covered with jungle.

It is capable of drifting along plains to a considerable distance from its source, particularly in the direction of the prevailing wind. It ascends mountains, especially when favoured by ravines and currents of air. The height to which it can ascend from its source is still matter of dispute. Dr. Parkes thinks that 500 feet is the limit in temperate climates, and from 1,000 to 1,500 in tropical countries; while others maintain that in the latter we are not safe from its influence until a height of 5,000 feet has been reached. It is probable that when men suffer from malaria at elevations above 2,000 feet, it is either derived from unsuspected local sources, or it is carried up ravines by currents of heated air from the unhealthy plains.

It is a common belief in India that water is capable of absorbing malaria, and that periodic fevers, dysentery, and even cholera, are produced by drinking water so charged.

This absorbing power of water, and especially salt-water, has often a beneficial effect, when a sufficient breadth of it, not less than from three-quarters of a mile to a mile, is interposed between our habitations and the source of the poison, which is either absorbed or rendered innocuous in its transit.

Belts of trees interposed in like manner exercise a protective influence.

Malaria disappears before cultivation and subsoil drainage, with free exposure of the soil to the action of the air and of living vegetation. When, however, the cultivating hand of man is withdrawn and the old conditions reappear, malaria again resumes its sway.

It is the cause of intermittent and remittent fevers, and their sequels; it "underlies" the cause of dysentery and cholera; and by its depraving influence on the constitution it often silently undermines the health without the manifestation of any febrile phenomena. Major-General Cotton, in his evidence before the Indian Sanitary Commission, very truly observes, "that there are many ailments which the natives of India call fever, but which a medical man does not, which are the effects of malaria."

When a person has for some time suffered from the toxic influence of miasm, a curious impress of periodicity is sure to show itself in all his subsequent ailments, whatever be their nature; and I believe, from extensive observation, that this impress of periodicity is never eradicated.

Casorati, a late Italian physician of eminence, in his "Treatise on Intermittent Fevers," a posthumous work recently published, has given it as his opinion "that miasm is the cause of an extremely small number of intermittent fevers." He says "that there are pernicious intermittents, the origin of which is simply rheumatic." Casorati further dwells on the fact that, within the sphere of his observation, "nothing is more common than to see pregnant women the subjects of tertian fever, under which they frequently abort;" and he gives numerous examples of diseases, such as menorrhagia, cephalalgia, &c. &c., all presenting an intermitting type, due, as Casorati supposes, not to the toxic effect of miasm, but to other causes, such as "humidity," "cold," and the like.

The truth is, that Casorati's sphere of clinical observation was in a malarial region: the stamp of periodicity was therefore deeply impressed on a great number of the diseases that came under his care. The proof of this is not far to seek; for, by his own showing, no treatment was effective until quinine was given. We do not find, where there is no miasm to complicate the case, that "acute rheumatism" or "menorrhagia" or "cephalalgia" derive benefit from anti-periodic remedies, still less that such are indispensable to all treatment.

No sooner is the blood poisoned by malaria than it acts on the stomach and alimentary canal. In all agues, particularly of a severe type, there is from the first great disturbance of the stomach, and in severe remittents this is often the most prominent and urgent symptom. Casorati goes so far as to state that morbid appearances in the stomach constitute by far the most constant post-mortem appearance found in fatal cases of intermittent fevers. In the article Dysentery I have given it as my opinion that miasm is also the cause of that disease. In the present state of knowledge, it is not possible to explain why malaria should in one case cause dysentery, and act with intensity on the glandular structures and mucous membrane of the great intestine, and in another excite an intermittent or remittent fever, with signs of extreme irritation of the stomach and duodenum, going on often to structural changes in those parts. Chemistry may one day reveal to us some difference, at present inappreciable, in the constitution of miasmata to account for the affinities displayed in the different cases.[1]

The structural changes of a more secondary kind induced by malaria are, enlargement of the spleen and liver, to be more particularly described further on.

INTERMITTENT FEVER.

Definition.—A specific paroxysmal fever, the febrile phenomena observing a regular succession, characterised by a cold, a hot, and a sweating stage, followed by a period of complete apyrexia, varying in duration according to the type of the fever.

Synonyms.—Periodic Fever, Ague, Paludal Fever.

History and Modes of Commencement.—When the human system has been exposed to the influence of malaria, sooner or later, according to circumstances, symptoms of disturbance appear: as already remarked, many have their constitutions silently undermined without suffering from periodical fever at all. It seems probable that in such cases the poison is not presented to the system in a very concentrated form; the blood is so gradually changed that the organs become as it were tolerant of its presence, to such an extent at least that febrile phenomena are not excited at regular intervals for the apparent purpose of expelling it from the blood. On the other hand, people in perfect health may be exposed to the action of malaria in such a noxious form as to be at once completely overwhelmed by it. The late Lieutenant-General Sir Mark Cubbon informed me that many years ago, when on a journey to the Neilgherry Hills, he was compelled to pass a night at the foot of the Segoor Pass, then an uncleared and unhealthy spot. A party of three German missionaries were also detained at the same place, and slept in the same house. These gentlemen were fresh from Europe, and in high health. On the following morning they pursued their journey, and were soon "above fever range." In less than twenty-four hours three out of the four of the party were stricken with fever, and two of them died in a few days.

Most frequently the person who has been exposed to malaria suffers for some days from premonitory symptoms. The toxic influence is evidenced by some degree of nausea and loss of appetite, with muscular pains in the back and lower limbs, with usually a slight feeling of chilliness, soon passing into trifling heat of skin, scarcely marked enough to excite attention. This may recur for several days before a regular paroxysm of ague sets in. Or, without such prolonged warnings, after an hour or two merely of the above symptoms, the patient may be seized with the cold stage, in the manner to be presently described. In such cases there is almost always a considerable amount of urinary irritation, the patient having frequent calls to pass pale-coloured, acid, and irritating urine. When this symptom is urgent, a severe paroxysm may usually be expected.

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[1] I am informed by Dr. E. Goodeve, late professor of medicine in the Calcutta Medical College, that the cases of dysentery which gave him most anxiety in Calcutta were those in which he was at first uncertain whether the disease was to be remittent fever or dysentery.

Then follow in succession the three stages which characterise this fever, viz. the cold, the hot, and the sweating stages, at the end of which there is a period of apyrexia, termed the *intermission*, the duration of which varies with the type of the fever. The time occupied by a paroxysm and the period of apyrexia that follows is somewhat incorrectly termed the *interval*. The types of the fever are named according to the length of the interval. These are the *quotidian*, which recurs daily, having an *interval*, in the above-mentioned acceptation of the term, of twenty-four hours; the *tertian*, with a paroxysm every other day, and an interval of forty-eight hours; the *quartan*, every third day, and an interval of seventy-two hours.

These are what have been termed the *regular* types of Intermittent Fever. Physicians recognise others which have been called *irregular*: such, for example, as the *double tertian*, which is said to differ from a quotidian only in having on alternate days fits corresponding in severity, character, and duration; the *triple tertian*, which has two fits on one day, and one the next; the *duplicated tertian*, which has two paroxysms on alternate days, with a fever-free day; the *double quartan*, which has a fit on one day, a mild one the next, the third being a fever-free day; and so on. What is it that determines whether the type of the attack shall be a quotidian, tertian, or quartan? It is probable that this is governed simply by the extent to which the blood has been charged by malaria. The presence of a quotidian seems to indicate a high degree of saturation, requiring a more frequently renewed effort of nature for at least its partial elimination than either a tertian or a quartan.

The tertian is said by many authors to be the primary type of fever, and to be the most common of all. This is certainly not the case in India, where without doubt the quotidian is the most common, and the quartan the rarest of all intermittents.

According to my experience in India and China, a first attack of ague invariably takes the quotidian form.

The duration of a paroxysm of Intermittent Fever varies with the type. It is longest in the quotidian, which lasts from eight to ten or even twelve hours; the tertian lasts from six to eight; and the quartan four to six hours.

The paroxysms do not always recur exactly at the same hour of the day. In the early days of an attack, when the disease, not having been interfered with by treatment, is "waxing," the cold stage will almost certainly appear an hour or two earlier on the days of the second and third paroxysms than on the first. On the other hand, when the system has been affected by antiperiodics, or the poison has by successive paroxysms been to some extent eliminated, and the disease is "waning," the time of attack will be postponed for an hour or two. I have observed this in my own person, and have noted it in others as of almost invariable occurrence.

The length of time to which intermittents left untreated will run on will depend much on climate, locality, and season, and the extent to which the system has been charged by malaria. Mild quotidians often terminate after ten or twelve paroxysms; quartans last longer, and may run on for months.

When once the system has gone through the phenomena of an attack of ague, paroxysms are liable to recur quite irrespective of fresh exposure to malaria; an error in diet, exposure to wet or cold, any cause that disturbs the balance of the circulation, may bring on an attack, and an impress of periodicity is apt to be given to any ailment from which the person may subsequently suffer. This disposition lasts always for years, sometimes for life.

The type of the disease does not always remain the same: a quotidian may pass into a tertian or a quartan; and an intermittent may, under certain conditions, assume the more grave form of a remittent.

I have repeatedly known sportsmen in India, and officers of the Forest Conservancy department, whose amusements or occupations exposed them only to mild intermittents so long as they remained in comparatively cool and elevated regions, suffer from severe remittent fever on descending to the plains, a change in many instances apparently due merely to the influence of high temperature, for I have several times observed it when there was no reason to suppose that the sufferers had been exposed anew to the influence of malaria.

The direct mortality from Intermittent Fevers in India is small. Even in Bengal, out of a strength of 344,152, with 111,687 admissions, the percentage of deaths to strength is 0·24, and the percentage of deaths to admissions is 0·76.

But although it is undoubtedly a rare thing to see a person die in the course of an uncomplicated Intermittent Fever, it is nevertheless, indirectly, an exceedingly destructive disease; the fatal results must however be looked for under other heads in the death returns of malarial regions. It is undoubtedly true, as remarked by the Indian Sanitary Commissioners, "that diseases of important organs, the consequences of malarial fevers, occasion much of the subsequent sickness, mortality, and invaliding among British troops serving in India."

Judging from the writings of Casorati and others, Intermittent Fevers seem to be more severe and more fatal in Italy than in India. How far the system of treatment in that country influences the mortality, I am not prepared to say. In many cases of simple, and apparently in all complicated agues, Casorati not only highly extols blood-letting, but reprobates its neglect as culpable and dangerous in a high degree. For example, in a case of ague with orchitis, Casorati draws a pound of blood, and does not hesitate to repeat the proceeding; and in all cases where gastric irritation, headache of extraordinary severity, and such like symptoms are present, his treatment is "decidedly antiphlogistic," comprising repeated blood-letting, both general and local, with perfect abstinence from food for many days. In Italy malarial fevers that in the opinion of physicians there demand such treatment are termed "pernicious." In India practitioners of the present day would be disposed to transfer the term from the fever to the treatment, for it is certain, as demonstrated by the experience of the past, that in any of the types of the malarial fevers of tropical regions antiphlogistic treatment such as that urged by the modern Italian school, is not only unnecessary, but most dangerous.

CAUSES.—Whatever tends to depress the physical or mental powers, and so render the system more liable to the influence of malaria, is a *predisposing cause.*

The *exciting cause* is undoubtedly an exhalation from the soil, given off under the conditions already described, to which the name of malaria is provisionally applied. That specific agues ever arise from other causes than malaria I do not believe, and am satisfied that where they are attributed to "cold," to "moisture," to "irritation," to the "influence of the mind," and such like supposed causes, it will in every such instance be found that the sufferer has at some former period been in a malarial locality, or that this poison, arising it may be from an unexpected source, has been in operation just before the attack.

In a most especial manner I desire to express my entire dissent from the doctrine that specific agues are the result of suppressed cutaneous secretions, under sudden impressions of cold: if it were so, we should have agues constantly occurring in temperate climates during the summer months, in places where no miasm exists, which is contrary to all experience. Where this poison has been introduced into the system, suppressed cutaneous secretion under sudden impressions of cold may call the poison sooner into action, perhaps by concentrating it more in the gastro-duodenal mucous membrane during the state of congestion that follows the impression of cold; but to produce a true specific ague I believe the presence of malaria in the blood to be necessary.

To the question, why the miasmatic poison, unlike that of rheumatism, or variola, or typhus, should produce a periodical, and not a continued fever, no satisfactory answer has yet been given; notwithstanding all the ingenious speculations of scientific inquirers, it remains unexplained.

SYMPTOMS.—After certain *premonitory* symptoms—of which the most prominent are nausea, languor, lassitude, muscular pains in the back and legs—the *cold stage* commences. In this the patient becomes chilly, first in the extremities, then in the back, and soon passes into a most unpleasant sensation of coldness all over the body. The skin shrivels, the nails become blue, and rigors, more or less severe, rapidly succeed each other. In the hottest climate the patient demands to have bed-clothes heaped upon him, although he derives little additional warmth from them. With the above there is often urinary irritation, the patient passing at short intervals considerable quantities of highly acid urine almost devoid of pigment.

This symptom, although not commonly mentioned by authors, I have very often experienced in my own person, and have frequently noticed it in others; when present in a high degree it adds much to the patient's distress.

The sensation of cold of which the sufferer complains is merely a subjective symptom. (Parkes.) Incredulous as the shivering patient may be, it is certain that the temperature of his blood, even before the rigors begin, is above the natural standard, a fact which is at once demonstrated by placing a thermometer in his axilla, which rises rapidly until it indicates a temperature of 105° to 106·3° Fahr. (Parkes, Ringer, Wunderlich.)

This sudden rise in temperature is common to all the types of malarial fever. Headache is sometimes complained of, but not always; the mind is inert, and occasionally the patient is drowsy. The duration of the cold stage is variable; it may last from half-an-hour to two hours and a half, and in rare cases even three or four hours. When the paroxysms have been often repeated, and the poison has been, to a great extent, eliminated, the cold stage shortens, until at last the patient is only conscious of a passing chill.

Hot stage.—Flushes of heat at first alternate with slight rigors. By-and-by a grateful feeling of warmth steals over the body; the bed-clothes are thrown off; the increase of temperature is now apparent to the patient and his attendants, without the aid of a thermometer. The pulse becomes full and frequent; the respiration, although still hurried, becomes more regular. When the hot stage is fully developed, a temperature of 107° or 108° is often noted. The agreeable sensations that accompanied the first feelings of warmth pass away; nausea, and even vomiting, often distress the patient; headache and thirst are complained of; and the patient tosses uneasily in a burning fever.

Physicians in the malarial parts of the Southern States of America look anxiously for the development of what they call "a good hot stage," regarding powerful reaction as conducive to the patient's safety; whereas a quick and feeble pulse, with rapid thoracic respiration and low temperature, are looked on as dangerous symptoms, as indeed they are.

The duration of the hot stage is usually about two hours; in severe cases it may last four or five, and it has been known to be prolonged through ten or twelve hours.

Sweating stage.—Perspiration appears first on the brow and face, and gradually spreads over the entire surface, until the patient sweats copiously at every pore. The pulse falls in frequency and strength;

the respiration becomes more natural; the temperature rapidly falls to the normal standard; headache first abates, and then passes away.

Captain Burton, the renowned African traveller, writing of the mild Intermittent or Seasoning Fever of East Africa, declares "that there is nothing unpleasant in these attacks. The excitement of the nerves is like the intoxication produced by a plentiful supply of green tea; the brain becomes uncommonly active, peopled with a host of visions; and the imagination is raised almost to Parnassus." This mental excitement I have experienced, and the observant traveller is right when he adds, "the patient pays for it when the fit passes off. These agreeable sensations do not recur with the subsequent paroxysms."

During the intermission the patient is commonly said to be "well," but this is only true in a limited sense; and if the paroxysms be allowed to go on unchecked, the sufferer, even during the intermission, soon becomes incapable of much exertion of mind or body.

Condition of the Urine.—As already mentioned, the urine is increased during both the cold and hot stages, and apparently, from the presence of a large quantity of free acid, is sometimes very irritating. Convalescence is ushered in by a remarkable diminution in its quantity; it now becomes scanty, alkaline, or neutral, and of a deep orange colour. Intelligent patients soon learn to note this, and intimate to their physicians the occurrence of what they deem a critical discharge, by informing them that no more quinine is required. In the hottest weather, during the active stages of intermittent fever, urine always retains its acid reaction for several days. When the fever intermits, the urine then rapidly undergoes decomposition, and changes from acid to alkaline. (Jones.)

A person with ague, not actually suffering from a fit, secretes less urea than a person in health. The moment a fit commences, the urea suddenly increases, although every known cause of increase, as food and exercise, be avoided.

The increase lasts during the cold and hot stages, and then sinks, sometimes gradually, sometimes suddenly, through the sweating stage, or into the commencement of the intermittence. The amount then falls below the healthy average. (Parkes.)

There is a very close connexion between the temperature and the amount of urea. (Ringer.) The amount of urea corresponding to a degree of Fahrenheit is greater at a high than a low temperature.

The pigment is lessened in amount. (Jones.) Uric acid is greatly increased during the fit. (Parkes.) The chloride of sodium, according to Professor Ringer, is greatly increased during the cold and hot stages. Albumen is found in an uncertain proportion of cases during the fit, with blood and renal cylinders. I can confirm, from personal observations, Dr. Parkes' remark that chronic Bright's disease is a consequence of ague. Many "old Indians," who have suffered from malarial fevers, die of this disease.

The Blood is changed from the beginning of the attack, and, probably, for some time before. The red globules and fibrin are diminished;

the coagulum is larger and more flabby than that of healthy blood, much darker in colour—in extreme cases approaching to black—and on exposure to air, instead of the usual bright red, it only assumes a cherry red colour. (Jones.) Its serum is dark and muddy, and it has sometimes an oily appearance.

The skin, after a time, assumes a dirty pale yellowish hue, a change which is often permanent, and which depends, not on bilious discolouration, but on some of the blood-changes above described.

Sufferers from Intermittent Fever are usually depressed in spirits, and are incapable of much exertion of mind and body; their appetites and digestion are bad, and they are prone to diarrhœa from slight causes.

When this anæmic condition is developed, a peculiar cardiac murmur is commonly present, which is prolonged into the great vessels. This is an "anæmic bruit," due apparently to the watery condition of the blood.

If the spleen is much enlarged, the heart is apt to be displaced upwards, and thus to mislead the unwary into a diagnosis of heart disease, when that organ is sound. (Morehead.) The heat-generating power of all victims to malaria is impaired : hence they suffer from atmospheric changes, of which healthy men take no note.

DIAGNOSIS.—Remittent fever is the only disease which appears to me likely to be confounded with an ague. The regularity of the phenomena, the existence of a distinct period of complete apyrexia, will suffice to determine the diagnosis, and in doubtful cases the thermometer will settle the point, "for all the types of ague present this characteristic peculiarity of a sudden and speedy rise of the temperature up to 105° or 106° of Fahr. and of an equally rapid and complete defervescence, till the period of another paroxysm comes about." (Aitken.)

MORBID ANATOMY.—Death in an uncomplicated intermittent is so uncommon that few opportunities for post-mortem examination are afforded.

We have seen that one of the earliest indications of disturbed function after the action of malaria is given by the stomach ; in the mildest agues this disturbance is present, and in severe remittents intense nausea and urgent vomiting are among the most prominent symptoms. Casorati, who has had many opportunities of dissecting the bodies of those who have died from the "pernicious" agues of Italy, observed "that sufficiently well-characterised morbid appearances in the stomach constitute by far the most constant post-mortem appearance of all those observed." (*Vide* "British and Foreign Med. Chir. Rev." July, 1864.)

Hyperæmia of the stomach and duodenum, then, is one of the most common of the appearances found *post mortem* in intermittents. In some of the cases examined after death at Walcheren circular ulcers, according to Sir Gilbert Blane, were found in addition to the hyperæmia above described.

The liver and spleen also suffer, the latter more frequently than the former. In recent cases the spleen is generally found so softened in its texture as to break up under examination; occasionally it is reduced to a dark-coloured bloody pulp, enveloped by its capsule. In more chronic cases the organ is found to be indurated, and often so enormously enlarged as to extend downwards into the pelvis. That the spleen acts as a diverticulum in the cold stage of ague there is no doubt; by percussion we can demonstrate that it enlarges with every fit, and contracts again when the paroxysm comes to an end. In time the elasticity of its structure is impaired, and some degree of permanent enlargement results. But there is another cause in operation. Virchow and others have shown, that the spleen enlarges not only in intermittent and typhoid fevers, but also in most other morbid processes resulting from the presence of noxious matters in the blood. Irritation of the gland ensues, and the result is increased cell-formation in its structure.

The liver is found in recent cases in various states of congestion, often soft in texture, and of a dark purple or black colour. In more chronic cases it is enlarged, the malarial poison acting as a source of irritation, leading, as in the spleen, to increased cell-formation. When the bodies of men who have served long in malarial regions are examined, one of the most common appearances is a deposition of black pigment in the spleen, liver, and kidneys.

PROGNOSIS.—I have never seen a person die from uncomplicated Intermittent Fever.

The prognosis in such cases, under rational management, is favourable.

The danger to life is from the malarial cachexia, and the organic changes to which it gives rise. When the disease is about to yield, not only are the paroxysms less severe, but the time of accession is postponed for some hours. The appearance of a copious deposit in the urine, and an herpetic eruption about the lips, are also favourable signs: so also are the disappearance of præcordial distress, anorexia, and nausea at the commencement of the paroxysm.

TREATMENT.—"Happily for us," says the observant traveller Burton, "the old African treatment is now obsolete. A. B. caught fever —gave him calomel, bled him, blistered him—died on the third day." Happily too for those whose lot is cast in India, the same may be said; the antiphlogistic treatment of malarial fevers is no more heard of there. So completely is this the case that it seems to me like contending with a shadow to say a word in condemnation of it. In Italy, however, this system not only holds its ground, but appears to be carried out with a higher hand than in the darkest days of African practice. Every perversion of normal function occurring in the course of an ague is still looked on as an inflammation, and treated accordingly by general and local bleedings, and a rigorous system of diet. What Dr. Haldane has recently said on this system of treatment generally is, as it appears to me, peculiarly applicable to every form of malarial fever that has come under my observation. "Formerly, when an inflammation manifested itself, it was regarded as something superimposed upon the organism; as an enemy attacking the fortress of life, which required to be attacked by the most energetic measures. Its supplies must be cut off by the enforcement of a rigorous diet, and it must be attacked with the heavy artillery of bleeding, mercury, and blisters. But it was not kept in mind that by these measures the garrison was weakened in an equal degree with the enemy, or rather in a greater degree, so that even if the adversary were overcome and retired from the contest, the patient often succumbed, owing rather to the severity of the treatment than to the malignity of the disease." (The Modern Practice of Medicine: a Lecture by Dr. Rutherford Haldane, M.D. F.R.C.P.)

Most systematic authors direct us to begin the treatment of all fevers with an emetic. In Intermittents, when the tongue is very foul or the stomach oppressed by food, an emetic does good, giving great relief, and hastening the stage of reaction. The longest cold stage I ever saw was not in India but in England. The sufferer was an Indian officer who was subject to ague. He had partaken freely of pickled oysters; in a few hours he was seized with intense nausea, headache, and epigastric oppression, soon followed by severe rigors. When I saw him he had been nearly five hours in this state, and his condition caused great alarm. I immediately gave him a mustard emetic, which speedily relieved him, and rapidly brought on a short and mild hot stage.

It is hardly necessary to caution even young practitioners against the absurdity of giving an emetic to a patient every time he has an ague, without regard to the special circumstances of his case.

The same rule applies to purgatives. They are useful if the bowels are loaded; the action of a purgative tends to relieve the congested condition of the solid abdominal viscera, and prepares the way for the action of quinine. But great caution is required in the use of purgatives in persons labouring under malarial cachexia, particularly in Asiatics.

If there be much urinary irritation, a few grains of bicarbonate of potash with or without a few drops of tincture of opium will relieve it at once.

During the cold stage the patient should have a sufficiency of bedclothes, but, beyond this, interference is not often called for, and most sufferers prefer to be let alone. If the cold stage is unusually protracted, or it becomes apparent that the vital powers are so oppressed as to be unequal to the development of reaction, then the external application of warmth and the use of stimulants may be required.

In like manner during the hot stage little interference is called for. The bed-clothes should be removed as reaction advances, cooling drinks may be given if they are relished or called for, which I have observed is seldom. If the patient be in a debilitated state from any cause, instructions should be given to watch the patient when the sweating stage begins, lest symptoms of collapse should appear, in which case support and stimulants should be promptly given.

The paroxysm ended, our utmost efforts should be directed to counteract the poison, and prevent, if possible, a repetition of the attack. In cases of first attacks it is of unspeakable moment to the patient to prevent his system from getting, so to speak, into the habit of going through the phenomena of an ague fit. For, although we cannot doubt that the aim and end of the process is so far salutary that it rids the system of a portion of the poison, it is also true that the organism suffers in the process, and that every paroxysm is a step towards the establishment of those organic changes I have described. In quinine, skilfully used, we have a remedy, particularly in first attacks, which almost deserves the epithet "divine," which has been applied to it.

I always assume that in first attacks the type of the disease will prove to be quotidian, and I take my measures accordingly, making sure that the patient shall have thirty grains of quinine between the termination of one paroxysm and the hour when we may look for another. The first dose of ten grains should be given towards the close of the sweating stage, and looking to the fact that when an attack is, as I have already expressed it, waxing, we may expect the setting in of the cold stage perhaps two hours earlier than on the first day, the last ten grain dose should be given so as to anticipate that time by at least an hour. Quinine in the treatment of agues should always, if possible, be given in solution, with a few drops of diluted sulphuric acid; its bitterness is best covered by the addition of a little syrup of orange-peel.

If irritability of stomach be present to such an extent as to lead to vomiting, no time should be lost in giving the quinine by enema. The lower bowel must first be washed out with a little warm water, and fifteen grains should then be given by the rectum in four ounces of beef-tea, if that be at hand, if not, in a little thin starch; and the irritability of stomach still continuing, this should be repeated twice at proper intervals during the apyrexia. I have been in the habit of administering quinine in this way with advantage for the last fifteen years.

If we are fortunate enough to prevent the recurrence of the paroxysm in this our first attempt, the gain to our patient is great, and it is well to maintain a moderate degree of cinchonism for some days, evidenced by "ringing in the ears." This can be effected by giving three or four grains of quinine in solution every four hours. But our duty to the patient is not yet discharged. We must not dismiss him from our thoughts because we have successfully managed his case so far.

In a lunar month from the date of his first attack, even should he not in the interval be exposed to malaria afresh, there will be a tendency in his system to repeat the same phenomena as before, and this tendency will be strengthened by every successive attack. A day or two, then, before the time, the patient should again be brought under the influence of quinine, which should be maintained until that time is past.

In military and naval practice we have great facilities for carrying out this prophylactic plan, by simply keeping the names of the men in a list, and requiring their attendance at the hospital for a few minutes daily at the proper time.

Speaking from a large experience, I promise those who may try it the happiest results, not only to the men themselves, but to the State, whose costly servants they are.

When our object has been attained, and the paroxysms have been broken, it is well, if quinine excites nausea or irritability of stomach, to give what may still be required after food, which will entirely obviate any such unpleasant effect; and, what is of no small consequence, prevent the patient from being disgusted with the remedy.

If we fail in entirely checking the return of the paroxysm, we are nevertheless pretty certain to have made some impression, and it will be a favourable sign if it is postponed for two or more hours, as is almost certain to be the case. Under such circumstances we must proceed as before, slightly increasing the dose of quinine, if we are not satisfied with the extent to which cinchonism was induced.

Most American authors describe quinine as the *antidote* to the poison of malaria. According to Herapath's experiments, not much of the quinine taken into the system is excreted from it, at all events when disease is present. Out of forty grains given to a man with tetanus, only a fifth part was detected in the urine; the remaining four-fifths were either assimilated in the body, or destroyed in their transit through the vascular system.

When given in ague it does not appear so soon in the urine as in health. (Parkes; Ringer.) "It has no marked effect on the water, urea, and chloride of sodium, though it may at once arrest the rise of temperature;" and Dr. Parkes adds, "After ague has been apparently cured by quinine, there occurs in the next two or three days an increase in urea, chlorine, and water, at the hours when the fit would have occurred but for the quinine. In other words, the quinine dissociates these two symptoms, increased temperature and ureal increase; it stops the first at once, but not the second for some days."

We occasionally meet with cases in which quinine appears to have lost its control over the malarial poison, the paroxysms returning with unfailing regularity month after month, in spite of the regular prophylactic use of the "*antidote.*" In such cases it will be found that the sufferers have been long in a malarious locality; and, whether or not we can detect enlargement of liver or spleen by palpation or percussion, the miasmatic and melancholic aspect of the patients, and the presence of bile in the urine, point to the imperfect manner in which the hepatic functions are being performed. In such cases a course of the fluid extract of taraxacum with small doses of podophyline are most useful; and if to the above be added the free use for some days of such blood depurants as the bicarbonate, or acetate, or citrate of potash, considerably diluted, it will be found that quinine, before useless, will soon reassert its power. I have seen this again and again, and by this method have cured agues deemed beyond the reach of art without a change of climate. I have no space to enter into the question of the *modus operandi* of such remedies. It is probable that they act chemically on the effete matters in the blood, which in some way interfere with the due action of quinine on the *materies morbi.*

Next to quinine as a therapeutic agent in this disease comes arsenic. It has been used in the East in the cure of agues and their sequels from remote antiquity. Having always been fortunate enough to have access to an abundant supply of quinine, I have not used arsenic much in the treatment of agues. From motives of economy it is much used in the French army, and in much larger doses than British physicians are in the habit of prescribing. Boudin, acting on the principle that in paludal fevers there is great tolerance of arsenious acid, is in the habit of giving it in divided doses, and, with the watchfulness always required in the use of this powerful poison, to the extent of a grain and even a grain and a half in the intermission. (Morehead, *Clinical Researches.*) Like quinine, its use should be continued for some time after the cessation of the fever, of course in diminished doses. We should carefully look for the earliest signs of its constitutional action; these are watering of the mouth, a silvery appearance of the tongue, redness of the eyes: and the medicine, as a rule, should be given after food. In the brow-ache and hemicrania of malarial localities arsenious acid is very efficacious—often more so than quinine.

I gave an extensive and careful trial to sulphate of bibeerine, prepared by Messrs. Duncan and Flockhart of Edinburgh, and found it useless in the treatment of all forms of Intermittent Fever. Barberry, a very old remedy in ague, I have also tried in the shape of tincture. I think it possesses some power as a febrifuge, but it is so uncertain in its action, so immeasurably inferior to quinine, that, in my judgment, to use barberry when quinine is available, is to trifle with the constitutions of our patients. Of Warburg's Tincture, as a febrifuge of undoubted efficacy, I shall have to speak when I come to remittent fever.

Diet.—I have already shown that Captain Burton has sound therapeutic notions on malarial fevers; I think him equally sound in what he says on diet. He observes: "People will act up to the old nursery saying, 'Starve a fever, feed a cold.' My experience in East Africa long ago untaught me that tenet. I have ever since preferred to support exhausted nature with essence of meat and beef-tea, and, when such things are procurable, with champagne, brandy cum soda, and y° oldest hock in y° cellar." These may not be available for hospital purposes; but when such wines are used, they should be "the best in the cellar," or they should not be used at all. Dr. Blair, of Guiana, in his admirable treatise on Yellow Fever, is quite as explicit on this point as Captain Burton. In the adynamic forms of intermittent, or in any form occurring in asthenic subjects, due attention to support during the intermission is a most essential part of the treatment, and it has been very well insisted on by Dr. Morehead, in his great work.

Treatment of Complications.—In the course of an Intermittent Fever, cerebral, pulmonary, hepatic, and gastric complications may occasionally be expected. After no small experience I unhesitatingly say, that the occurrence of drowsiness, mental confusion, suffusion of countenance, and such like symptoms, should not mislead us into the use of routine remedies directed against them. Let all our efforts be used to cure the fever, to stop the paroxysms, and to the due support of our patients. When these objects are attained, the head symptoms will disappear. In like manner the presence of cough or asthma, or of hepatic congestion, should never induce us to lay aside quinine and the other means recommended above, in order to direct routine treatment to counteract this or that incidental symptom, occurring in the course of a malarial fever. Those who do so often put the lives of their patients in great peril.

Practitioners who omit the use of quinine from a groundless fear of aggravating such symptoms, and substitute strong measures of a so-called antiphlogistic kind, applying leeches and cold to the head, giving nauseating expectorants and active purgatives, merely because of the head symptoms already mentioned, the presence of some bronchitic râles and cough, or some tumefaction of the side, will not only have little success in the treatment of malarial fevers, but will aggravate the complications they seek to cure, hasten alarming exhaustion, or bring on sudden collapse.

I have seen epilepsy, long dormant, developed again during the course of a severe Intermittent Fever. The same principle guided me in the management of this case; I redoubled my efforts to extinguish the fever with quinine, and succeeded, and with the fever the epileptic attacks disappeared. Had I interrupted the administration of quinine and directed my efforts against the nervous complication, I feel assured that I should have made no impression on it. With each recurring paroxysm, the epileptic attack would have been repeated, the old habit would have been re-established, and, if nothing worse happened, my patient would have become a confirmed epileptic. It is an old observation that nervous diseases are sometimes remedied or superseded by Intermittent Fever. One case, and one only, lending some support to this supposed fact, has come under my observation. A medical officer in India became subject to epileptic attacks, which resisted treatment. It was feared that he had become a confirmed epileptic. He was attacked by Intermittent Fever, which affected him at intervals for many years; but from the date of his first paroxysm of ague he never had another epileptic attack. I knew this officer in India well; the fits of epilepsy were genuine, and the attacks severe and frequent.

Treatment of Malarial Cachexia.—The first and most essential part of the treatment is removal to a non-malarious climate. If for this end a sea voyage be possible, so much the better; but unless the ship on which the voyage is made be one in which strict hygienic discipline is maintained, much of the good to be expected from this measure will be lost; and if, from inattention to proper dietetic rules, a scorbutic taint be added, the condition of the patient will be seriously aggravated. I have dwelt on this at some length in the *Army Medical Report* for the year 1861-62.

A nutritious diet, abundance of exercise, pure air, and pure water, are powerful curative means. The careful use of some of the means practised by the so-called "hydropathists" for restoring the functions of the skin is strongly to be recommended; a walking tour on the mountains of Scotland, or, better still, a season or two on the moors, will do much to work the poison of malaria out of the system. Great attention should be paid to clothing, which should be warm, particularly on first coming into high latitudes. Sir Ranald Martin has well shown the evil consequences resulting from want of attention to this.

Returns of Intermittent Fever should be met by the prophylactic use of quinine, so given as not to excite gastric irritability. If the patient's appetite be bad, he may take a course of syrup of the phosphate of iron, quinine, and strychnine: a formula for this preparation (now much used at the R. V. Hospital, Netley) is given below,[1] for which I am indebted to my colleague, Dr. Aitken. Some of the other preparations of iron may be, after a time, substituted; a very suitable one is the tincture of the acetate, from the old Dublin Pharmacopœia.

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[1] The following is Dr. Easton's formula for the preparation of the syrup of the phosphates of iron, quinia, and strychnia (syrupus ferri, quiniæ, et strychniæ phosphati):—

"℞. Ferri Sulph., ʒv.; Sodæ Phosph., ʒvi.; Quiniæ Sulph., grs. cxcii.; Acid. Sulph. Dil., q.s.; Aquæ Ammoniæ, q.s.; Strychniæ, grs. vi.; Acid. Phosph. Dil., ℥xiv.; Sacchar. Alb., ℥xiv.

"Dissolve the sulphate of iron in one ounce of boiling water, and the phosphate of soda in two ounces of boiling water. Mix the solutions, and wash the precipitated phosphate of iron till the washings are tasteless. With sufficient diluted sulphuric acid, dissolve the sulphate of quinia in two ounces of water. Precipitate the quinia with ammonia water, and carefully wash it. Dissolve the phosphate of iron and the quinia thus obtained, as also the strychnia, in the diluted phosphoric acid; then add the sugar, and dissolve the whole, and mix without heat.

"The above syrup contains about one grain phosphate of iron, one grain phosphate of quinia, and one thirty-second of a grain of phosphate of strychnia in each drachm. The amount of phosphate of quinia might be increased according to circumstances, and if eight grains of strychnia were employed in place of six, as in the above, the phosphate of strychnia would be in the proportion of the one twenty-fourth of a grain in every fluid drachm of the syrup. I would scarcely venture on a much larger dose. In cases of delicate children, with pale countenances and deficient appetites, I have given, with great benefit, a combination of equal parts of the above syrup and of that prepared by Mr. Edward Parrish, often called "chemical food." To children between two and five years of age the dose of this combination may be a tea-spoonful three times daily." (*Vide* Aitken's Science and Practice of Medicine, vol. ii. 657.)

Treatment of Enlarged Spleen.—This "ductless vascular gland" enlarges, as has been already stated, under the influence of malaria, without, as well as with, the intervention of fever. The treatment advised above, exercising, as it is calculated to do, a favourable influence on the constitution of the blood, often acts beneficially also on the enlarged spleen. Should this organ, however, continue undiminished in size, we have a powerful remedy in the ointment of the biniodide of mercury, applied over the gland. The experience of many practitioners in India having demonstrated the extraordinary efficacy of this remedy in cases of goitre, it has of late years been successfully used in some parts of India as a remedy in solid enlargements of the spleen.

When used in goitre, a portion about the size of a nutmeg is applied over the swelling with a smooth spatula, the patient is directed to expose his goitre to the sun's rays as long as he can bear the smarting which quickly follows. A second application is immediately made, and it rarely happens that any further treatment is necessary.

Within the last six months we have been very successful in the R. V. Hospital, Netley, in treating enlarged malarial spleens and livers with this ointment. I have pursued the same plan, substituting the heat of the fire for that of an Indian sun. In some cases, where the spleen has extended down into the pelvis, it has, after several applications, been reduced almost to its normal limits; and in only a few cases has it failed to reduce the size of the organ most signally. It has acted just as energetically on enlarged malarial livers.

The mode of treatment is worthy of extensive trial, and I am the more induced to recommend it to the notice of the profession in England because in not a single example has it induced any unpleasant constitutional action.

I am so impressed with the unsuitableness of mercurialisation in cases of mercurial poisoning, that I have watched narrowly for any ill effects from the use of this compound, without observing anything to justify my fears; on the contrary, in every case where it has acted on the spleen or liver in the manner above described, an immediate improvement in the patient's general health has been the invariable result.

REMITTENT FEVER.

Definition.—A specific paroxysmal fever, with exacerbations and remissions, characterised by a slight and ill-defined cold stage, which does not recur at every exacerbation; an intense hot stage, with violent headache and gastric irritation; and an almost imperceptible sweating stage, which is sometimes wanting.

SYNONYMS.—Bilious Fever, Bilious Remittent, Endemic Fever, Marsh Remittent, Gastric Malarious Remittent, Jungle Fever.

HISTORY.—This, the gravest form of true miasmatic fevers, has been observed wherever malaria is generated in sufficient concentration, both in hot and in temperate climates, but it is most prevalent and fatal when high temperature and malaria act in combination.

Our armies have suffered from it in Spain and Portugal, at Walcheren, and in the Mediterranean; it prevails with great intensity on the western shores of Africa, and in the course of its great rivers. In the East Indies it is most common as well as fatal in the delta of the Ganges, in the terrais, in the jungles at the base of mountain ranges, and, at certain seasons, in many uncultivated and jungly plains.

It is found in America, North and South, prevailing chiefly, with varying degrees of severity, in the vast region between the northern lakes and the Gulf of Mexico, and it is a formidable endemic in the West India Islands.

Remittent Fever is a more serious disease than any type of intermittent, and the direct mortality from it is much higher.

In all intermittent fevers there is a period of complete apyrexia; in remittents, the defervescence is not complete. The more urgent symptoms between one exacerbation and another abate; in some cases this abatement is well marked, in others it is so slight that the period of so-called remission may escape the notice of all but a wary and experienced observer.

A remittent fever may, after a time, pass into one or other of the types of an Intermittent; and, conversely, an intermittent may assume the graver form of remittent, either under the influence of a fresh charge of malaria, or, as I have frequently observed, under the stimulus merely of exposure to a higher temperature.

Remittent Fever, as we have seen, has many synonyms, but there is one, the use of which I earnestly deprecate, viz. Inflammatory Remittent, still used by many authors, although not always in the same sense. Some employ the term merely to denote a high degree of intensity in the febrile phenomena, without wishing to imply the existence of inflammatory action; while others use it in the belief that the terrible disturbance of the vascular and nervous systems is due to a genuine phlogosis. In either view the term is objectionable; one is based on false views of the true pathology of the disease, and both are calculated to mislead in treatment.

Modes of Commencement.—As in all paludal Fevers, so in this, symptoms of gastric irritation are the first evidence of approaching disorder. The patient complains of præcordial anxiety, anorexia, and nausea, with weariness, langour, and lassitude. Uneasiness, sometimes amounting to great oppression, at the epigastrium, is perhaps the most constant, as it certainly is the most distressing, of the signs of an approaching attack of remittent fever; it is sometimes present for twenty-four or thirty-six hours before the setting in of the cold stage.

The time of day at which the first paroxysm takes place is not uniform, but when the disease is established a morning remission is almost an invariable rule. (Wood; Hunter; Morehead; Balfour.) It is a point of great practical importance to observe in each case the periods of exacerbation and remission, and the duration of each. If the exacerbation begins at noon, it will usually decline about midnight, or a little before, and the remission will last till noon next day. Such cases are usually comparatively slight and manageable. Or the exacerbation may begin at midnight, continue all night, and remit in the morning, the remission lasting till midnight. Or, in severe cases, there may be a double exacerbation, at noon and at midnight, the remissions being in the evening and morning, the last being invariably the most distinct.

Where the disease assumes this type, the evening remission is sometimes so slight as to escape notice; but for the invariable morning remission it might be mistaken for a continued Fever. Treatment will of course have an effect on the periods of exacerbation and remission, just as it has on the paroxysms of an Intermittent.

Duration.—This may be stated at from five to fourteen days. Like all forms of miasmatic fevers, the duration of the disease is much affected by treatment and the action of antiperiodic remedies.

Modes of Termination.—The Fever terminates in recovery, or by passing into some type of intermittent, or in death. When the disease ends in recovery, as it usually does, the fever generally culminates in a profuse perspiration, and so ends. Sometimes the amendment is gradual, the exacerbations become less severe, the heat of skin is less pungent, the vomiting and oppression at the epigastrium subside, the pulse diminishes in force and frequency, the tongue becomes moist and clean, the remissions are longer and more distinct, and they are ushered in by free action of the skin; and in this gradual way the Fever subsides.

In some remittents, in which the paroxysmal character has been from the first well marked, if antiperiodic remedies have not been skilfully used, the disease is apt to become chronic, and to pass into one or other of the intermittent types.

I have never seen death take place earlier than the eighth day. This event appears to be determined by the destruction of the vitality of the blood by the agency of the morbific cause.

When death occurs at a later period, the patient sinks, partly from the cause just mentioned, and partly from exhaustion, consequent on the excessive and prolonged excitement of the system. Here again treatment exerts a powerful influence, and may determine not only the time but the mode of death.

Cause.—On this subject I have nothing to add to what has already been said under the head of intermittent fever. I believe that all forms of remittent fever are due to the same miasmatic cause as intermittent aided probably by high temperature. When a Remittent follows exposure to malaria, it is reasonable to suppose that the morbific agent has been absorbed in a high degree of concentration.

I attribute some effect to the agency of high temperature, because remittents are more common in hot malarious countries than where the same poison exists in temperate regions, and because, as already mentioned, I have, in a great many instances, observed intermittents pass at once into a severe type of the remittent form, apparently from no other cause than the descent from comparatively cool and elevated regions into the heated plains, and this under circumstances when there was no reason to believe that the sufferers had been again exposed to malaria.

The period of incubation is also, I think, much influenced by temperature—being shorter in hot, longer in cold climates—and doubtless also by the degree of concentration of the poison. I have already given an example of a fatal miasmatic fever being developed within a few hours after exposure to the cause in a notoriously malarial locality. From a week to ten days or a fortnight have in my experience been the most common periods of incubation.

SYMPTOMS.—These in an uncomplicated Remittent Fever, occurring in an adult of a tolerably vigorous constitution, are as follows: The most constant, as well as the most urgent, of the premonitory symptoms, already described, is oppression at the epigastrium. The cold stage is neither so complete nor so long continued as in ague; in some of the worst remittents I have ever seen, no rigors were observed at all, a passing sensation of chilliness, alternating with flushes of heat, being all that the patients complained of, as regards temperature. In other cases rigors are present, but they are seldom severe; here, as in ague, these sensations of cold are merely subjective, the thermometer indicating already a temperature perhaps 2° above the natural standard, which, as the hot stage develops, mounts to 7° or 8°, and, according to some observers, even 10° above that standard.

As the hot stage advances, vomiting often begins, and continues throughout the disease a distressing and embarrassing symptom. The sense of fulness and oppression at the epigastrium is not relieved by the vomiting, although the amount of fluid thrown off is out of all proportion to the quantity taken. The tongue is furred, and, as the temperature of the body rises, dry. The pulse, which in the premonitory stage was slow, small, and irregular, now rises rapidly to 100 or 120; in men of sthenic habits it is full; in adynamic cases from the first, although very frequent, it is small and compressible.

The countenance is flushed, the eyes suffused, and the patient complains of rending headache, with pains in the limbs and loins; the skin is red and distended; the heat ardent and stinging; the sufferer is restless, and tosses in his bed in the vain search for an easy posture.

When the above symptoms have lasted for from six to twelve hours, they begin to abate: a slight degree of moisture breaks out on the brow and neck, and gradually spreads over the body; the pulse comes down in force and frequency; the heat of skin diminishes; there is some relief of headache; vomiting ceases; and the patient obtains some sleep.

This is the period of remission.

In severe fevers it is sometimes hardly possible to distinguish any but the morning remission, so slight is the abatement. Keeping this in mind, it should be anxiously looked for. The pulse will often give the only indication, and the physician who knows how precious for treatment such golden moments of remission are, will diligently watch for the earliest signs of lull in the midst of this tempestuous action of the system.

After a respite of some hours, varying from two hours to eight or twelve—seldom so long—the fever returns, often without a chill, or so slight a one as hardly to attract notice; all the symptoms above described are renewed, and always in an aggravated form. This is technically called the exacerbation, which in due time passes into the remission again.

I have already pointed out the usual course of the periods of remission and exacerbation, but I repeat once more that the attack is dangerous just in proportion as the disease resembles a continued and not a paroxysmal fever.

Of all the symptoms *nausea* and *vomiting* are the most constant and the most exhausting: the vomited matters at first consist of any food that may be in the stomach, then of a watery fluid, often in surprising quantity. Soon bilious regurgitation takes place into the stomach, and the rejected matters become of a greenish yellow colour, then brown, and finally, in extreme cases, black, resembling the "black vomit" of yellow fever. The resemblance will be more striking if, as sometimes happens, the skin assumes a yellow tinge, and a hæmorrhagic tendency be evinced. I have seen two cases at Madras, both in officers of the Forest Conservancy department, in which the hæmorrhagic range was most extensive, the patients passing blood from the stomach, bowels, and kidneys.

In one of these cases I had the advantage of the assistance of Dr. Cornish, the able Secretary of the Principal Inspector-General of the Madras Army, and we both agreed that the symptoms in this case came nearer in their *ensemble* to those of true yellow fever, than any we had ever seen in the whole range of our experience in the east.

Headache is a prominent symptom. It is described as throbbing at first, passing, in subsequent paroxysms, into a constant pain with some degree of tension of the brow, sometimes said to be "rending."

Delirium.—Some confusion of thought is common enough, but violent delirium is rare. In very adynamic cases, or where depletion has been freely used, low muttering delirium precedes the coma in which cases of this description often close.

Hiccough is often troublesome where gastric irritability is severe, but it usually appears as the disease is passing off. In one of the cases alluded to above, it lasted for days.

The Bowels are usually constipated in the first instance, sometimes large watery evacuations appear early in the disease, at other times when it is passing away.

Practitioners whose choicest weapon against "bilious" remittents is calomel, are but too familiar with the dark brownish black evacuations of cadaverous odour, the appearance of which too surely indicates that an unfavourable termination of the case is at hand.

The state of the *urine* is deserving of the closest study.

Many authors of great authority write of it as always scanty, high coloured, and of high specific gravity. But in severe Indian Remittents I have noticed the opposite condition. Dr. Cornish, I am sure, must remember that in the case we saw together there was even diuresis throughout, until convalescence was established. I have notes of three other cases, in which the same condition prevailed; in all four the urine was bloody. It is invariably acid, seldom contains albumen, so seldom that Dr. Parkes has proposed this as one of his points in the diagnosis between yellow fever and malarious Remittent, on the strength of Ballot's observation, who found the urine albuminous in 300 cases of yellow fever. According to Jones of Georgia, the urea is increased in severe remittent fever; and the uric acid lessened till convalescence, when it increases. The pigment also is lessened. (Parkes on Urine.)

The older authors describe, and very graphic some of their descriptions are, what they called *putrid* Remittents. These, as Sir Ranald Martin has well shown, were all complicated with scurvy, the inevitable result of protracted sea voyages to India in overcrowded and otherwise unsanitary transports, not supplied with antiscorbutic remedies. Soldiers treated in this way were landed on the malarious shores of Bengal, and when smitten with the endemic Fevers of the country, the symptoms presented "putrid" phenomena, and the mortality was shocking. The medical officers of the first expedition to China in the year 1840, had an opportunity of observing, under the unsanitary arrangements which characterised so conspicuously the first occupation of Chusan, the terrible effects of malaria and scorbutus combined. An entire regiment, 900 strong, was almost destroyed by malarious Fevers and bowel complaints in a few weeks, and such of us as survive can bear testimony to the truthfulness of the description of "putrid" remittent fevers, given by the writers above alluded to.

Without, however, any scorbutic taint, we may have Remittent Fever presenting from the commencement an adynamic character. I was very familiar with cases of this kind when serving in the immediate vicinity of the great native city of Hyderabad in the Deccan. From the malarial quarters of that densely populated and most unsanitary city, I used to receive into the Residency Hospital, during the autumnal months, a number of cases of this kind, presenting from the first, signs of great depression, the fever after the second or third exacerbation becoming almost continued, the skin being yellowish and covered with petechiæ, the pulse exceeding 120, small, and compressible, the tongue dry and black, the teeth covered with sordes, the respiration quick, and sometimes irregular, the abdomen distended, the bowels loose, and a disposition to hæmorrhage from nose, mouth, and bowels, and almost invariably delirium, with a tendency to coma. Such cases, unless energetically treated, hasten rapidly to a fatal termination by exhaustion and coma.

Remittent Fever with *cerebral affection* is usually met with in India in sthenic Europeans lately arrived in the country, who have been living imprudently and at the same time exposing themselves to the sun.

In such cases the exacerbation is severe with ardent fever, a full and firm pulse, mental confusion, and even delirium.

Hepatitis is a rare complication. This is Morehead's experience; in twenty-two years' service in India I never saw a genuine case of this complication. I have repeatedly alluded to gastro-duodenal irritation as a prominent symptom in malarial fevers; tenderness, proceeding from this cause, is often mistaken for hepatic inflammation.

Jaundice.—Some degree of yellowness of the skin is common in remittents, both of an ardent and adynamic type; but completely developed jaundice is more rare.

It occurred in twenty-eight out of 114 of Dr. Morehead's select clinical cases; I have seen a few examples of this complication, but never had an opportunity of examining one after death. Ten of Morehead's cases proved fatal.

Pressure on the common biliary duct by the head of the pancreas was found in one; in another the hepatic and common ducts were obstructed by a lumbricus; and in two there was constriction of the cystic duct. Traces of inflammation of the mucous membrane of the stomach and duodenum were observed in six cases. In almost all there was enlargement of the lymphatic glands situated near the entrance of the common biliary duct into the duodenum, but not to such an extent as to cause pressure. (Morehead.)

Splenic enlargement is found, as in other types of malarial fever, but not so frequently as in Intermittents, probably because the cold stage in remittents is not so prolonged, and also because the whole duration of a remittent is shorter than that of an intermittent fever. When found after death it may, as in some of Morehead's clinical cases, be traced to the influence of previous attacks of an intermitting character.

Diagnosis.—After what has been said it is unnecessary to enter into the points of difference between an intermittent and a remittent fever.

There can be no doubt that enteric fever and the adynamic forms of remittent have often been confounded in India, and yet the diagnosis, to a careful observer, is not difficult. In the former, if the characteristic eruption appears, it will settle the point; but, in India, this is more frequently absent than in this country. Gastric and duodenal irritability are present in all remittent fevers, absent in enteric.

The fever is more or less paroxysmal in the former, continued in the latter. In Remittent the skin is frequently yellowish; not so in enteric fever. In the former, apart from the epigastric and hepatic regions, abdominal tenderness is generally absent; present in the latter. In Remittent, the stools are dark coloured and bilious; in enteric fever, they are brownish yellow, with whitish yellow-coloured flocculi floating in them.

If hæmorrhagic tendency evinces itself in the course of a Remittent, the blood proceeds from the mouth, nose, urinary organs, and bowels; while in enteric fever, it is from the intestines only, at an advanced stage, during the separation of the glandular sloughs. Lastly, careful examination into the history of the case, with an eye to causation, will materially assist the diagnosis.

Specific yellow fever being unknown in India, no question as to diagnosis between it and Remittent Fever can arise there. Elsewhere, the diagnosis will be established, as it appears to me, by attention to the following points:—

Yellow fever is continued; all Malarial Fevers are paroxysmal.

Albuminous urine is the rule in yellow fever—a rare exception in remittent. The spleen enlarges during the paroxysm of malarial fever, but is not affected in the other. Hæmorrhage from the stomach and other viscera is the rule in yellow fever; comparatively rare in malarial fevers. Death is common in yellow fever on the 3rd day, but seldom occurs in the most malignant remittents before the 7th.

Over yellow fever quinine has no power, save when there is a malarial complication; over true malarial fevers of every type, its power is beyond the reach of question. Convalescence after yellow fever is rapid and agreeable; slow after malarial fevers.

PATHOLOGY.—Having gone fully into the pathology and *post mortem* appearances of Malarial Fevers in the previous article, it is unnecessary to enter again into a description which would involve much useless repetition; such points as more especially belong to Remittent Fevers I have incidentally considered in connexion with the symptoms.

PROGNOSIS.—Death from an uncomplicated Remittent Fever ought to be a rare occurrence, and, under good management, so it is. Even in ardent cases in sthenic constitutions, the prognosis is favourable; and, if skilfully treated, recovery may be confidently looked for, in a large majority even of adynamic cases, if seen sufficiently early.

The early subsidence of gastric irritability, headache, and frequency of pulse, are favourable signs. Distinct remissions, with action of the skin, and postponement of the exacerbation, are also evidence of an approaching favourable change. On the other hand, if the fever is more continued than paroxysmal, with a pulse failing in strength while it gains in frequency; if there be a tendency to collapse at the close of the exacerbation, profuse hæmorrhage from the stomach or bowels, cold sweats, delirium, and coma; in a word, signs of extreme exhaustion, these all point to danger, and indicate the necessity for restorative treatment of an energetic character.

TREATMENT.—The disease is not an inflammation; therefore, bleeding or antiphlogistic remedies have no legitimate sphere of action in the contest. I freely admit that a young physician has no more difficult lesson to learn on being brought face to face with his first case of tropical Remittent Fever, in the person of a young and vigorous adult, than to restrain his hands from the use of such means. When he feels the burning heat of skin and full pulse, sees the flushed face and congested eye, the tongue furred and dry, and listens to complaints of a head racked with pain, and limbs vainly seeking relief in incessant change of posture; and, in addition, sees his patient distressed by an intolerable sense of oppression at the stomach, which is not only not relieved, but is even aggravated by almost incessant vomiting;—when, I say, all this is seen for the first time, an inexperienced man is apt to say this is the "inflammatory remittent of so and so," "here antiphlogistic treatment must be used, or my patient is lost."

I believe there is another and a better line of treatment available.

The best possible hygienic arrangements should be made, and care taken to secure careful ventilation of the room or ward.

The history of the case should be inquired into, particularly with a view to ascertain the hour of first accession, so that the period of probable remission may, as nearly as possible, be calculated, in order that the visits of the attendant should be so timed as to insure the patient being seen, particularly at the morning remission.

Cold stage.—This, in Remittent Fever, is so transient that patients are seldom seen at this time, and no special treatment is required.

Hot stage.—It is seldom necessary to order an emetic in fulfilment of the time-honoured practice of beginning the treatment of all fevers with one. Vomiting, in a great many cases, requires rather to be checked than encouraged: where, however, there is much nausea and oppression, or sense of fulness at the epigastrium, without vomiting, a few tumblers of tepid water may be given, which will answer the purpose. It is well, also, that the bowels should be thoroughly cleared as soon as possible. For this purpose, from three to five grains of calomel, extract of colocynth, and scammony should be given, with a few drops of any aromatic oil. I have found this combination very effectual; it rarely, if ever, gripes or causes nausea; it appears to act on the whole tract of the intestine, and seldom requires a nauseous draught to aid its operation.

If the hot stage be mild, without much headache, ardent heat of skin, epigastric or hepatic tenderness, not much interference is required, beyond giving the patient small quantities at a time of iced or well-cooled water, soda-water, or lemonade, if any of these are relished.

If, however, the reaction be very vigorous in a sthenic adult, with severe headache, pungent heat of skin, severe pain in the loins, and much restlessness, something must be done for his relief. The hair should be cut close, and cold should be assiduously applied to the head, taking care that this is not done, as it often is, so as to act as a fomentation.

The inordinate heat of the skin may be reduced (*a*) by cold affusion; (*b*) by sponging the surface with tepid water; (*c*) by enveloping the body in a sheet, wrung out of cold water, and changed two or three times; (*d*) by packing the patient in the wet sheet, after the manner used in hydropathic establishments. Each method has its advocates; and in such cases as I have described above, I do not think there is risk in any of them. Where, however, there is marked epigastric or hepatic tenderness, with a tendency to extreme hepatic or splenic congestion, as pointed out by Morehead, the use of the wet sheet *alone* is objectionable, as tending to aggravate that condition. The wet sheet-packing acts powerfully on the skin, and is more likely to relieve, than to increase, congestion of internal organs. This method, however, must be used with caution, if at all, in cases of an adynamic character, as exhaustion and even collapse might follow the powerful sudorific action it generally excites. The sponging of the surface with tepid water is always safe and grateful to the patient.

Vomiting in severe and so-called "bilious" Remittents is often one of the most distressing symptoms. I have seen it induce great exhaustion. It is best combated by giving small quantities of ice-cold water, or by sucking small lumps of ice in the fold of a handkerchief, by the repeated application of sinapisms to the epigastrium, by inhaling the vapour of a *few drops* of chloroform, or by the addition of the same to an effervescing draught. I have seen the wet sheet-packing most effectual in restraining vomiting and relieving the distressing sense of oppression at the stomach, which the sufferer vainly endeavours to get rid of by incessant retching.

Hepatic and splenic tenderness should be relieved by fomentations, stupes, or sinapisms. Sometimes a pad of lint well sprinkled with chloroform covered with oiled silk, and applied to the epigastrium, is very soothing. By treatment of this kind, diligently used, the necessity for abstracting blood by leeches is obviated.

Treatment during the Remission.—On the first signs of this, as soon, that is, as moisture appears, and the heat of skin abates, and the pulse comes down in force and frequency, quinine should be given in an effective dose of not less than ten grains. I have given fifteen often, twenty sometimes; I am no advocate for excessive doses, and any quantity over twenty grains I deem to be excessive. I am never deterred from giving quinine merely because, even in the remission, there may be headache or foul tongue. Experience has taught me that the best remedy against those conditions is the one that acts on the toxic agent that is the primary cause of all the disturbance.

I am never deterred from giving quinine, because the remission is slight; so soon as I am satisfied that there *is* an abatement of symptoms I proceed to give the remedy, in the full belief that if I make a skilful use of these golden moments of remission, however slight this may be, I shall be rewarded at the end of the second exacerbation with one more distinct and perfect than the first; knowing also that if through timidity I suffer the first remission to pass unimproved, the next may be more faint, unsatisfactory, and difficult to recognise. I by no means wish to imply that there is always so much difficulty in recognising the remission. I desire only to guard the inexperienced against expecting to find the remission always so distinct as we find it described in books.

If quinine is rejected, and the irritability of the stomach is such that a second dose is also vomited, twenty grains ought at once to be administered, in any bland fluid, by enema. If the stomach retains the medicine, it should be repeated every second hour until thirty or thirty-five grains have been taken before the hour of expected exacerbation.

Suppose the purgative has not operated, are we to wait for its operation before giving quinine? I have done so, but finding that I lost more ground by delaying quinine than I gained by the action of the purgative, I abandoned the practice. At the same time I am fully alive to the necessity of securing free action of the bowels at as early a period as possible.

As soon as the second remission appears, quinine must be given as before, and continued until full saturation of the system is evidenced by cinchonism, or by a distinct abatement of the disease. Ringing in the ears and deafness are unequivocal signs of cinchonism. So soon as this state is brought about, in a vast majority of instances, the exacerbations will become milder, and terminate in a copious sweat, and the patient will enter on a state of convalescence.

Having ventured to write with so much confidence on quinine used in this way, I am glad to be able to adduce the testimony of Dr. Davy to the safety of the measure. This high authority says, "That in the Remittent Fevers of the West Indies, during the first quarter after the practice was introduced of giving quinine in full doses to cinchonism, out of 165 cases only two proved fatal, and the record of the post-mortem examination in the two fatal cases shows that they were rather instances of latent phlegmasiæ than of fever of the true remittent type."

To this unexceptionable testimony I shall only add the evidence of one other observer of equal reputation and experience. The late Dr. David Blair, Surgeon-General of British Guiana, thus expresses himself on the question of the safety of quinine:—"It has been prescribed by me to patients of both sexes and all ages, and, where ascertainable, almost invariably to cinchonism, during thirteen years, and probably to the extent of several thousand ounces of the sulphate, and during that time I have seen no danger from its effects, with the exception of three or four cases of imputed abortion."

During the remission the patient should have mild farinaceous diet, milk, chicken tea, and such like. As soon as the gastric irritability subsides, beef-tea should be given, and on the first sign of exhaustion, nourishment and stimulants should be given at short intervals.

Should quinine ever be given during the exacerbation? In the adynamic form of the disease, such as I described as coming from the malarial quarters of the city of Hyderabad, I never waited for a remission, but gave it at once by mouth or rectum, or both, combining with it the assiduous use of support and stimulants at short intervals.

The American physicians led the way in this practice, and demonstrated its safety.

Again, when called to cases which have been mismanaged in their early stages, either by the neglect of quinine, or the too free use of antiphlogistic means, we must act in the same way. There is no time to wait for a remission: the low form of exacerbation then present will hourly assume more and more a continued type; the remissions, if they appear, will be of short duration, and it will soon be hardly possible to recognise them at all. Such cases can only be saved by energetic means, by quinine, support, and stimulants, given in quantities regulated by their effects. In this way most hopeless-looking cases may be snatched from the jaws of death, as I have seen in a great many instances. What I wrote on the treatment of the complications of ague, I repeat here emphatically. Practitioners who relax in their efforts to stop the exacerbations, who pause in the use of quinine while they apply routine remedies for this or that symptom, now applying leeches to the head because delirium or headache is present, to the epigastrium because there is some tenderness there, will have little success in the treatment of the worst form of Indian Remittents.

My experience has satisfied me that such symptoms are most effectually met by the means which directly tend to counteract the poison which is keeping up the excitement, and disturbing the functions of the organs to which it is conveyed by the circulation; at the same time, active stimulation of the skin over affected organs should not be neglected. It is surprising how much relief may be given by sinapisms, turpentine-stupes, or stimulating embrocations, without having recourse to so double-edged a remedy as depletion.

Mercury has been largely used in the treatment of Remittent Fever. When I first went to India, calomel, chiefly in combination with James's Powder, was used to an extent that, to practitioners of the present day, is hardly credible. "Inflammation" was thought to play an active part in this fever, and as calomel, next to the lancet, was regarded as the most direct antiphlogistic remedy, it was used with the intention of subduing this inflammation.

The end sought was to "affect the mouth," and quinine, when given at all, was only ventured on when this desired consummation was attained, and then timidly and in utter ignorance of its real value and true therapeutic action. A practitioner of this school in India in the present day would be an object of terror to all educated men within reach of his prescriptions.

Beyond measure miserable is the spectacle of a man whose system already saturated with malaria, is still further depraved by the mercurial cachexy.

This was the system which, introduced into India by the late Dr. James Johnson and some of his followers, superseded the admirable treatment recommended by Lind, and others of his day. The consequences were not creditable to our art, and the return to more rational treatment, based on a sounder pathology, has been attended with a signal diminution in the mortality from all forms of malarial fevers.

Before concluding, I must add a few words on a febrifuge which, in Southern India at least, has attained a high reputation. I mean Warburg's Tincture.

This is a remedy the exact composition of which is kept a secret in the family of its inventor.

In common with the whole profession I deprecate the use of secret remedies, and, as a general rule, decline to use them.

But it is no secret that quinine enters largely into this combination, and is its most active ingredient. Whatever be the exact nature of its composition, I am bound to say that in Remittent Fever this tincture is, without doubt, an efficacious remedy. It is the most powerful sudorific I know. A person under its influence sweats until he saturates not merely his linen and bedding, but also the mattress on which he sleeps; and his breath and skin for days exhale a powerful and peculiar aromatic odour. I have seen it, in at least thirty cases treated by myself, cut short severe remittents after one or two exacerbations.

It has been largely used for many years by officers in the Mysore Commission, whose duties often expose them to fevers of a dangerous type, in the malarial districts of that province. Major-General Frederick Cotton, of the Madras Royal Engineers, in his evidence before the Indian Sanitary Commission, thus expressed himself regarding this remedy:—"Those engaged in opening the passes through the fatal jungles of the western coast of India found that medicine invaluable. In the Parambaddy Pass, especially, a trial of it was made on a very extensive scale, and with admirable results.

"When its efficacy became known, the coolies employed would work in the most dangerous parts of the forest, if they were sure that the medicine was at hand." The tincture is sold at a high price, in small bottles containing two doses. After the bowels have been opened by an aperient, one half a bottle is given, all drink is withheld, and at the end of three hours, the remainder is administered.

It has a fine golden yellow colour, apparently from tincture of aloes, and its taste is persistently bitter. Sometimes in three hours after the first dose, more frequently in an hour after the second, profuse diaphoresis sets in, and this goes on until the bedding is saturated.

In a great many cases there is no exacerbation after this sudorific action. So great is this action of the skin, and in some cases so exhausting, that this remedy, used in the manner above described, would be highly dangerous in adynamic cases—a fact not adverted to in the printed directions which accompany every bottle. If used at all in such cases, it should be in much smaller quantities than those ordered ; and support should be given as soon as the skin begins to act.

My friend Dr. Morehead speaks slightingly of this tincture, but after many trials of it, and a full knowledge of the success that has followed its use in many formidable types of malarial fever, I do not hesitate to recommend that careful trials should be made of this remedy in regions where these fevers prevail. But, I repeat, it requires caution ; if given indiscriminately in the manner recommended by Warburg, I am confident its use must often be disastrous.

DYSENTERY.

By Professor W. E. Maclean, M.D.

Definition.—A specific febrile disease, characterised by nervous depression; by inflammation and sloughing of the glandular apparatus of the mucous membrane of the large intestine (sometimes extending into the small gut); by tormina and tenesmus, with scanty, mucous, and bloody stools of a peculiar odour, changing, as the disease advances, to serous, and giving off a gangrenous effluvia.

Terminology.—*Δυσεντερια, δυς* difficulty, and *εντερον* an intestine; Dysenteria, Tormina, Fluxus Dysentericus, Colonia, Colonitis; Dysentery, Flux, Bloody Flux; Dyssenterie, Flux de sang, Fr.; die Ruhr, die rothe Ruhr, German; Dissenterie, Ital.; the Looseness (vulg.).

History.—Dysentery was well known to the most ancient writers on medicine, and has largely occupied the attention of modern physicians, particularly of those who have served in fleets and armies. It has been seen in all climates, in the temperate as well as in the torrid zone. No country has been exempt from it; sometimes appearing alone, at others as a formidable complication of malarial fevers or scurvy, often treading in the footsteps of devastating wars, for in all ages it has been the scourge of armies, and one of the chief causes of mortality in unsanitary camps and garrisons.

In the pre-sanitary age it was as common and nearly as destructive to human life in Britain as it is now in unhealthy tropical regions as yet unvisited by the sanitary reformer.

In Ireland "the looseness," as it was called, was a common endemic disease, causing great mortality throughout the seventeenth century, and prevailing occasionally in an epidemic form down to the year 1818.

Dysentery has ceased to be a destructive disease in this kingdom; it has disappeared before a higher civilization, and what it brings in its train, viz. improved agriculture and drainage, more particularly subsoil drainage, the removal of filth from the vicinity of dwellings, the supply of purer water to our cities, in a word, increased attention to hygiène. Just in proportion as Malaria, the product of moisture and organic decomposition in soils, has been banished from our midst, so has Dysentery ceased to be a prevalent and fatal disease.

In India, among Europeans of all classes, this disease comes next to fevers in frequency, but the direct mortality caused by it is greater than from all the forms of fever known in that country.

"Out of an aggregate British force of 25,433 men of Her Majesty's Army serving in periods of eight and ten years respectively in the stations of Calcutta, Chinsurah, and Berhampore, all in Bengal Proper, there occurred 8,499 cases of Dysentery and diarrhœa.—In the presidency of Madras, again, out of an aggregate British force of 82,342 men serving there from 1842 to 1848, there occurred 10,531 cases of Dysentery, and 9,189 cases of diarrhœa, making a total of 19,720 cases of bowel disease exclusive of cholera."[1] Nor is this all, for most of the casualties which occur amongst sick soldiers on the voyage homewards from India are from chronic Dysentery.

The Naval Medical Reports show that of late years, except on the East India, China, and West Coast of Africa Stations, bowel complaints do not cause a high mortality. In the Report for 1860 it is said that the "intractable flux" of China was, as usual, far more destructive of health and life than any other disease that attacked the force. The death-rate from this cause was in the ratio of 13·6, and the invaliding 25·9 per 1,000 of mean strength.

MODES OF COMMENCEMENT.—Dysentery in all its varieties commences in one or other of the following ways. After the presence for a longer or shorter time of signs of constitutional disturbance, the patient is severely griped, with frequent calls to stool, the motions becoming scanty, mucous, and bloody. Or, there is a burst of unhealthy semi-feculent biliary discharges, which soon give place to the characteristic stools first described. In either case the odour is offensive and peculiar, becoming gangrenous as death approaches, after extensive sloughing of the intestinal tissues.

The COURSE of the disease will depend on a variety of conditions, such as its type, whether it be mild or sthenic, malarial, typhoid, or scorbutic; also on the stage at which treatment has commenced, and still more on the nature of that treatment. In *mild acute Dysentery*, under favourable circumstances and rational treatment, the intestinal lesions are generally moderate in extent and degree; the duration of the attack seldom exceeds eight or ten days, sometimes not more than half that time; healthy alvine discharges, a copious lateritious sediment in the urine, and a free perspiration proclaiming restoration to health. Under less favourable circumstances, injudicious treatment, or, it may be, some imprudence on the part of the patient, the issue may be less fortunate and the case may pass into the chronic form. In *sthenic Dysentery*, the constitutional symptoms are more urgent, the local lesions are more severe, more rapid, more destructive, leading, if not checked by early treatment, to changes incompatible with life, or, after much destruction of the glandular and mucous tissues of the great intestine and much suffering, to an imperfect recovery. Still, this form of the disease, when early seen and well treated, gives a high ratio of speedy and complete recoveries. The duration of the disease is from nine days to three weeks. It may prove fatal on the eighth or ninth day, or death may not take place until the end of the third week; this termination is ushered in by sudden cessation of abdominal pain, increased fluidity, and gangrenous effluvia of the stools, and delirium.

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[1] Sir R. Martin.

In the *malarial form,* the course of the disease partakes of the periodic nature of the complication. There are remissions and exacerbations of the febrile symptoms, the solid viscera of the abdomen are apt to be deeply implicated, and hepatic, splenic, or renal symptoms may seriously complicate the case, and determine an unfavourable issue.

The *typhoid or malignant form,* often with a scorbutic taint grafted on it, is the most hopeless and fatal type of the disease. This is the form seen in unsanitary camps and besieged garrisons, in beaten armies after great privations, and sometimes in armies that have not sustained any military disaster, but have the misfortune to be commanded by generals who set at nought the laws of health, as was the case notably in the first wars in Burmah and China, and more conspicuously, in the Crimea.

In this form the lesions are not confined to the colon, but, passing the ileo-colic valve, extend into the small intestine. The disease runs its course in about fifteen days or less; nervous depression is extreme, and the tendency to death is by exhaustion.

CAUSES.—When we consider the variety of causes to which this disease has been attributed, it is impossible to admit that agents so many and various can give rise to an affection which in all climates has presented so much uniformity in its symptoms and anatomical lesions.

It appears to me that many of the so-called "causes" of Dysentery must be regarded more as active agents of *propagation* than of *causation.* For my own part, I believe Dysentery to be caused by the action on the blood of a poison having a peculiar affinity for the glandular structures of the large intestine. This poison I believe to be a malaria generated in the soil by the decomposition of organic matter. Once a common and fatal disease in this country, it is now so rare that a London hospital physician rarely, if ever, sees a case of genuine specific Dysentery, save such as have been imported from malarious countries. How comes it that a disease with which our predecessors were so familiar has become so rare? Many of the commonly received "causes" are as much in operation now as then; *e.g.* the combined action of cold and moisture, the action of irritants on the mucous membrane, unripe fruit, unwholesome and indigestible food of all kinds, feculent and other accumulations in the larger intestines, yet Dysentery does not result. Is it not that, for the reasons already assigned, less malaria is evolved from the soil?

It seems that just in proportion as we have banished malaria, so have we got rid of Dysentery. For a long time the prisoners in the Penitentiary at Millbank were subject to visitations of Dysentery at those seasons, and in those states of atmosphere, which most favour the decomposition of organic matter in the soil.

The late Dr. Baly, then physician to the prison, in the Gulstonian Lectures for 1847, has given a most instructive account of an outbreak of this kind. Dr. Baly has shown that the disease which prevailed in Millbank prison was precisely the same in its symptoms, course, and lesions, as that described by Sydenham, and by writers on tropical diseases of the present time. He investigated its cause with much care, and was led to the conclusion that it was "due to a poison introduced from without, viz. a malaria rising from the soil," and that all the conditions required for its production abounded in the close vicinity of the prison.

In India, Dysentery prevails most and is most fatal in moist alluvial soils containing organic matter in a state of decomposition, as for example, very notably, in Calcutta.

It is no doubt true that the disease is sometimes seen, and that in a malignant form, in places which are not alluvial. I cannot give a better example of this than the old infantry barracks at Secunderabad in the Deccan, of dysenteric notoriety; but there, as at Millbank, the conditions necessary for the production of malaria were only too abundant.

The sad but instructive history of those barracks has been given by Staff-Surgeon Crawford and the writer of this article, in the Army Sanitary Report for 1860. The barracks stand (for notwithstanding their dreadful history they are still in use) on low ground, swampy on one side, and overshadowed by rocks on the other, exposed to the malarious influences of the marsh in the south-west monsoon, while the rocks on the other side shut out the invigorating north-east wind. A grave-yard, now closed, is placed close to the buildings, on a higher level, and in the direction of the natural drainage; another, at a greater distance and in a less objectionable site, is on the south side. The surface and subsoil are thoroughly saturated with organic matter, the removal of which is impracticable. The neighbouring soil until recently, when something has been done to fill them up, was furrowed by ravines, in which ordure was deposited by natives; the privy accommodation was of the worst possible construction and badly placed, the barracks were invariably overcrowded, and surrounded by a high wall. Here were all the conditions for the production of *malaria*, and the causation and propagation of disease.

For half a century the loss of life in these buildings, *chiefly from malignant Dysentery*, was shocking. For some years it was nearly one in three of *strength*; so late as 1826 it was nearly one in every five. Between the years 1837 and 1858, out of an annual strength of 834·44 occupying those buildings, the admissions into hospital were 1529·40, and the deaths 37·20. In 1858 the "Royals" occupied the barracks, with an average strength of 1,098; there were 2,497 admissions into hospital and 104 deaths, being nearly ten per cent. of the strength.

Here we have two examples, one in England the other in India, of the local existence of malaria with a like result, the production of malignant Dysentery; the symptoms and anatomical lesions, being alike in both cases, with this exception, that in the barracks, hepatic complications were common, due probably to the greater intensity of the cause, combined with high temperature, and intemperance among the soldiers. It is quite true that the provisional term malaria is a vague one. In the present state of knowledge we cannot isolate it, and must here take it to mean a poison resulting from the decay of organic matter in the soil, which, when conveyed into the body, is capable of causing a disease, of which certain anatomical lesions of the great intestine are a characteristic and invariable incident.

The following are usually named as "exciting" causes, but, as already remarked, it is more than probable that some of them are merely active agents in propagating a disease that has a specific cause.

Impure Water.—"There is," says Dr. Chevers, "the strongest reason for believing that much of the cholera and Dysentery which occur on board the vessels in the port of Calcutta is caused by drinking the always muddy and filthy and often brackish water taken up in buckets over the ship's side. Nearly every person, native or European, who comes to Calcutta suffers, more or less, just at first, from some kind of bowel complaint, but none suffer so much as seafaring men;" and no wonder; for the same authority informs us, "that opposite Calcutta the water is frightfully impure. There it receives some forty tons of excreta daily"—(and we may confidently assume that this enormous mass of impurity contains no inconsiderable proportion of cholera and Dysentery stools, for these diseases are always present in that most unsanitary city)—" a multitude of bodies of dead cattle, and some 15,000 corpses yearly." Dr. Rose makes precisely similar observations as regards seamen frequenting the port of Shanghai in China, and attributes the heavy mortality among them from Dysentery to the same cause, viz. drinking river water loaded with organic impurity, and still further polluted by the excrements of an immense population.

Miss Nightingale, in her summary of the Indian Sanitary Report, observes, with too much truth, "there is no reason to hope that any station (in India) has what in this country would be called a pure water-supply, and at some it is to be feared that, when men drink water, they drink cholera with it," and we may add, Dysentery also.

Exposure to Cold.—Dr. Mackay, R.N., describes the mode in which this cause operates among seamen serving in the malarious rivers of

China. The men, when they lie down on the deck to sleep, pull up their frocks and coarse under flannel jackets, so as to expose the abdomen. When the cool night wind sets in, the exposed skin of the sleepers, from being bathed in perspiration, becomes dry and finally chilled, and in a very short time they awaken griped, and perhaps sick, and so commences very frequently an attack of what Dr. Mackay calls "Sporadic Dysentery."

Impure Air.—Dysentery once established is propagated by the effluvia from the evacuations of those affected. In most Indian barracks a few years ago, the latrines were so badly constructed, so injudiciously placed, and so ill-kept, as to aid materially in propagating both Dysentery and cholera, by exposing the healthy to the effluvia arising from the evacuations of those affected. I affirm from frequent observation, that barrack-rooms most exposed to the effluvia of latrines always furnish the largest number of dysenteric cases, and the heaviest mortality.

In like manner I have seen the disease propagated in hospitals by the practice of preserving the evacuations of large numbers of dysenteric patients, for the inspection of medical officers at morning and evening visit. No single measure of a preventive kind yet tried has exercised a more beneficial effect on the health of troops in India, than the improvement which has been introduced in the position, construction, and conservancy of barrack and hospital latrines.

SYMPTOMS.—These will of course vary with the type of the disease. It is not pretended that in practice we can always expect to find the line separating one type of Dysentery from another to be sharply defined. The distinctions are not, however, fanciful, or adopted here merely for the sake of convenience in description; for those who have seen much of the disease in tropical countries must be familiar with the different forms about to be described. One form, it is true, often passes into another by insensible gradations, and a strong family likeness runs through them all.

Mild Dysentery.—Careless exposure to cold night air, or to a chill after exertion, is, in malarious regions, the most common exciting cause of this form of the complaint. The chill is succeeded by slight heat of skin, loss of appetite, and sometimes nausea. Griping pains in the belly, technically termed *tormina*, follow, with frequent calls to stool, the evacuations consisting of semi-feculent mucus, with or without an admixture of blood, passed with painful straining, called *tenesmus*. There is seldom much abdominal tenderness on pressure. The tongue is white and moist, thirst is not urgent, but even where the appetite is not much impaired, the griping which soon follows all but the mildest articles of diet makes the patient averse to solid nourishment.

The course, duration, and termination of every form of Dysentery are so much influenced by the patient's prudence or the reverse, and by the treatment to which he is subjected, that it is not easy to describe them. If he is reasonably prudent, abstaining from stimulants, unsuitable food, and the use of irritating purgatives, the disease will probably subside in from seven to fourteen days: under good treatment I have seen it do so in forty-eight hours, or less. The skin resumes its action, and the more this is the case the more rapid and complete is the restoration to health. Feculent evacuations return, griping and straining cease, and the urine deposits a copious red sediment.

It is not always that the disease runs so happy a course, and has such a fortunate termination. Too often young and inexperienced sufferers seek delusive ease from stimulants, strong port wine, or brandy, or both, while the more ignorant and needy try to quench their sufferings in the poisonous and fiery spirits of the nearest bazaar. Under such management the disease may soon assume a more formidable aspect, passing into the acute form, to be presently described, or, after a longer or shorter period of suffering, varying according to circumstances, it may become chronic.

Acute Dysentery.—The disease begins with a chill or well-marked rigor, soon followed by more or less heat of skin, with a quick but usually compressible pulse, and nervous depression. Tormina is much more urgent than in the mild form described above, and the calls to stool are more frequent and pressing. At first the evacuations may be semi-feculent and watery, but this state does not last long; soon they become scanty, muciform, and bloody. If the rectum is much implicated, tenesmus will be a distressing symptom. If the seat of the disease be higher up, not only will tenesmus be less urgent, but the nature of the stools will be different. In the latter case, depraved biliary secretions will be more abundant, and will be intimately blended with mucus, epithelium, and blood. If the rectum be deeply affected, the bladder will often sympathise, being either irritable or so paralysed as to require the use of a catheter, while the stools will be more muciform and the blood less intimately incorporated with them.

From the first the stools are very offensive; they give out an odour which is peculiar to Dysentery, an odour justly said by Dr. Parkes to be "the most offensive of all the organic effluvia." It is difficult to describe it, but once experienced it is never forgotten, and is in itself conclusive evidence as to the nature of the disease; and not only so, but an experienced physician can in some degree form an opinion from it as to the nature and extent of the mischief going on in the tissues of the affected bowel. The absence of tenesmus, although a great relief to the patient, is by no means *per se* evidence of a slight amount of disease; it merely indicates that the rectum is less involved than other parts of the colon.

Soon the calls to stool become more urgent and frequent, the patient is hardly in bed ere he desires to rise again, each time convinced that he is about to pass something that will relieve him. The abdomen becomes tumid, and, in the region over the parts most implicated, tender. Often this tenderness is well marked at one point and absent in others. At last the patient can hardly be induced to leave the close stool; he desires to remain on it, and strains involuntarily. His patience and temper give way, he becomes irritable, nervous depression increases, and the countenance indicates suffering and despondency. The stools, which at first consisted of a little feculence with bloody mucus, now contain portions of shreddy, granular exudative matter, often resembling washed raw meat. The disease still advancing, the countenance becomes more anxious and depressed; irregular febrile paroxysms come on; the pulse rises in frequency, while it diminishes in force; the abdomen grows more tympanitic; the tongue, still continuing foul in the centre, becomes red at the edges and dry, then dark brown, and finally black. If no improvement takes place, another change in the stools follows; they become serous, of a brownish colour and very copious, exhausting the patient terribly. The effluvia become insupportably fœtid, even cadaverous, pervading the whole ward, and penetrating into neighbouring rooms, unless the nursing is good. The abdomen becomes more tumid, but pain ceases. In this state the patient often flatters himself that all is well, deeming the cessation of pain a favourable symptom; by and by his mind begins to wander, and, as if in some degree to compensate for past sufferings, his delirium takes pleasing forms, and he dies exhausted without more pain. Under happier circumstances and good treatment, amendment begins before destruction of the affected tissues has gone to such a degree as to be incompatible with life, The stools improve, become feculent, and lose the dysenteric odour; blood and mucus disappear, the abdomen becomes less tumid and tender, the countenance improves (a most favourable sign), the febrile paroxysms disappear, the pulse gains in volume and loses in frequency, and the state of nervous depression passes away. There is still a third termination, the case may pass into the chronic stage; or this state may result after convalescence from the acute attack has been well advanced, in consequence of some imprudence on the part of the patient or those about him; errors in diet; or, in military life, from exposure to hardships before health has been sufficiently restored.

Chronic Dysentery.—As the patient sinks into this unhappy condition, he continues to lose flesh. The discharges, still maintaining much of the dysenteric odour, are for the most part fluid, but they vary from day to day, and even on the same day, being muciform, serous, and bloody. Sometimes they are tolerably natural, and even formed; more frequently they consist of thin feculence, of a reddish brown colour and most offensive smell. Often they are pale and frothy, and in this condition are usually voided with violence. Power over the sphincter is impaired, often lost entirely. The sufferer does not gain flesh, the appetite may be tolerably good, or capricious, or bad. In any case the food is hurried along the intestine, and the patient is poorly nourished; the tongue is red and glazed, sometimes deeply fissured; night sweats are frequent, the hair drops off, boils are common in various parts of the body, and the patient looks older than he is. This is the general condition, but it is liable to be modified by any complication that may be present, the malarious, or syphilitic, or scorbutic cachexia, or by hepatic disease. The condition of the patient will also depend on the extent of injury done in the acute stage to the mucous and glandular structures of the intestine, the presence or not of unhealed ulcers, the condition of the intestine as regards thickening of its coats, or the opposite state of atrophy; whether or not the lesions are bounded by the ileo-colic valve, or pass into the small intestine; and, in no small degree, on the extent to which the solid viscera, liver, spleen and kidneys are implicated.

Thus it will be seen that chronic Dysentery is not, as many suppose, merely due to abortive cicatrization of the ulcers left by the acute stage, but is often a much more complex state.

I believe, and shall presently demonstrate, that Acute Dysentery is in a high degree amenable to early and judicious treatment. On the other hand, speaking from a large experience, I affirm that complete restoration to health *by the unaided efforts of nature,* is an extremely rare occurrence; one of two things happens, either the disease destroys the patient, or it passes into the chronic form just described.

Malarious Dysentery.—By this term I mean to indicate those cases in which *malaria* acts with a high degree of intensity on the system, developing, in addition to the symptoms already described, those which are characteristic of malarious fevers.

It is in this form that we find hepatic complications most frequently present—complications which add greatly to the gravity of the disease, and unfavourably influence the prognosis: such cases will be recognised by the periodicity of the febrile paroxysms, the presence of gastric irritability, such as we see in remittent fevers, and by the peculiar nature of the evacuations, which from the first are serous, and contain little blood, but have the characteristic dysenteric odour. There is also a therapeutic aid to diagnosis; ipecacuanha is not well borne, and quinine acts powerfully in checking the febrile paroxysms.

Malignant Dysentery.—I have already described the conditions under which the disease is generated; it has been described as "Dysentery plus the typhoid condition." From the first, the patient has a cachectic aspect, the countenance soon becomes sunken, listless, and sodden, a burning heat in the belly is complained of, with great oppression and sinking; the voice is weak, the stomach irritable, and pulse frequent, small and compressible. The evacuations are serous and bloody, attended with tormina and tenesmus, and the dysenteric odour is intense from the beginning. For a short time after the first burst, the stools may be muciform and bloody, but this condition does not last long; they again become abundant, serous and bloody, consisting of dissolved blood, serum, epithelium and gangrenous portions of semi-dissolved mucous membrane, exhaling an insupportable gangrenous odour. The urine is scanty, scalding and fœtid, and in extreme cases the secretion is suppressed entirely, as in cholera. The skin is in general cold and clammy, and immense discharges of blood often take place from the bowels, determining the fatal issue, and also from the mouth and nostrils.

Scorbutic Dysentery is met with when the conditions which induce that cachexy are superadded to any type of Dysentery. I had an opportunity of seeing the ravages worked by this form of the disease during the first occupation of Chusan in 1840, one of the most disgraceful episodes in our military history. Here we have the usual signs of the scorbutic cachexy—pallor, emaciation, extreme lassitude, pains in the loins and limbs, a spongy condition of the gums, which bleed on the slightest pressure. Soon livid and purple-coloured spots appear on the legs, and ulcers of a weak and unhealthy kind follow the least injury to the surface, the pulse is feeble, the appetite bad, and the debility extreme. To the above are added the symptoms of Dysentery, only the evacuations are, from the first, fluid, bloody and nearly as offensive as in the malignant form. Scorbutic Dysentery does not run so rapid a course as the sthenic or malignant (typhoid) form, rarely proving fatal in less than three weeks, and, in the more chronic form, it may extend over as many months.

DIAGNOSIS.—This ought not to present any difficulty. In acute cases the tormina, tenesmus, muciform and bloody stools, and above all, their peculiar odour, will distinguish Dysentery from diarrhœa. If seen in a more advanced stage when the stools have become more fluid, the history of the case, the nervous depression, the blood in the discharges, and once more, their cadaveric odour, will establish the diagnosis.

The author once saw a case in which a grave error in diagnosis had been made. A lad between thirteen and fourteen years of age, the son of European parents in India, was seized with frequent calls to stool, with much gastric irritation, nausea, vomiting, restlessness, and oppression. The practitioner who first saw the case, seeing that the stools were scanty and muciform, without feculence, and passed with griping, pronounced the case to be one of Dysentery, and proceeded to treat it with calomel and opium. Under this treatment the stools, still presenting the same characteristic appearances, became of a greenish colour, and the gastric symptoms were aggravated. Seeing the patient at this stage, the first thing he noticed was the entire absence of the peculiar dysenteric odour in the evacuations. On inquiry, it was found that before the appearance of the so-called dysenteric symptoms, the patient, after slight febrile and catarrhal symptoms, had an eruption on his skin, which, after imprudent exposure to a cool sea-breeze, suddenly receded, the recession of the eruption being followed by the symptoms mistaken for those of Dysentery. The patient was placed at once in a hot bath; soon the characteristic rash of measles appeared, nausea and vomiting subsided. Under simple treatment the disease ran a mild course, and the lad soon recovered.

PATHOLOGY.—From what has already been said, it will be seen that I regard Dysentery to be as much the consequence of a specific poison as any of the recognised miasmatic diseases. Why this poison should, as Baly has expressed it, have such an affinity for the glandular structures of the large intestine, we can no more explain than we can tell why the poison of enteric fever should evince a like affinity for the glands of the small intestine.

Looking to the whole history of the disease, its modes of propagation and its characteristic lesions, it seems impossible to regard it as a simple inflammation of the mucous membrane, a mere colonitis, but rather as a specific disease, of which inflammation of the glandular structures and mucous membrane of the great gut, and its consequences, are characteristic and invariable incidents.

MORBID ANATOMY.—It is difficult to give a perfectly accurate description of the Morbid Anatomy of the complex structures involved in this disease. The descriptions of authors are at variance with one another. The confusion has arisen partly from the ignorance of some of the observers of the healthy structure of the parts described; and also, in some instances, from not tracing the change of structure from its earliest manifestations to its ultimate results, a fault due in many instances to lack of opportunity.

So great is the confusion in the descriptions, that my colleague Dr. Aitken has asked (1) "Whether distinct epidemics are characterised by distinct local lesions? (2) Whether two or more distinct diseases have not been confounded under the one name of Dysentery? Or (3) Whether the various local lesions described by different writers are only so many varieties, forms, or types of the same diseased process—a process modified in particular cases by constitutional peculiarities, or by other circumstances." This latter view Dr. Aitken is of opinion is the one most consistent with observation, and in accordance with what we know of the history of other miasmatic diseases.

In the Dysentery of temperate climates, it is seldom that more than one or two of the anatomical divisions of the great intestine are implicated. In tropical countries, not unfrequently, the morbid process affects the whole tract of the intestine from the cæcum to the anus. Except in Scorbutic Dysentery, it is rare to find the lesion extending into the small intestine.

I propose to describe the morbid appearances under the three heads of Congestion, Ulceration, and Exudation.

In the rare instances in which the affected bowel has been seen before ulceration of the glandular apparatus has commenced, it has been observed to be swollen and somewhat softened, the colour of the affected parts being rosy red, purplish, or of a brownish and sometimes a leaden hue. This discoloration of the mucous membrane is by no means uniform; in the non-febrile forms of the disease I have seen the membrane between the ulcers unchanged, and even paler than natural, and there is as much diversity in the degrees of softening of the tissues.

Ulceration.—It is mainly to the careful observations of Drs. Parkes and Baly that pathologists are indebted for an accurate description of this process. At the very commencement of the morbid action, Dr. Parkes has shown that the solitary glands are enlarged in various degrees, "from the size of a millet-seed to a small shot" (Baly). They are seen to be distended with a white exudation, some with a dark central spot, and all surrounded by a vascular ring. It is on and about these glands that the ulceration, or, as Baly prefers to call it, sloughing process, begins, spreading from them into the mucous membrane. Dr. Parkes was led from his dissections to the belief that ulceration almost always begins in the glands themselves, very rarely around them, and only occasionally, in very rapid cases, by effusion of fluid beneath the mucous membrane. This last is the "circumscribed sub-mucous suppuration," leading to the formation of circular and other shaped ulcers, described by Morehead, Haspel, and Bleeker. This commencement of the ulcerative process must be familiar to all who have seen the rapidly destructive type of Dysentery which so long prevailed in the old infantry barracks in Secunderabad, before described. The ulcers are at first circular and have rounded edges, but as they enlarge the edges become flat, and they spread out into irregular shapes, having for the most part their long diameter in the direction of the plicæ of the mucous membrane round the intestine. In the more advanced cases the whole tract of the gut is studded with ulcers of all shapes, sizes and degrees of development, presenting in different cases a great variety of appearances—some deep and irritable-looking, others more superficial and pale, some having their floors covered with lymph of various shades, others having their floors formed by the muscular coats. In the rapidly destructive sloughing Dysentery, I am of opinion that the process begins by sub-mucous purulent effusion, detaching the mucous membrane, which rapidly becomes gangrenous. I have seen the whole tract of the mucous membrane, from the rectum to the transverse colon, presenting a black gangrenous mass, in which it was impossible to distinguish a shred of healthy tissue.

As soon as the diseased action implicates the other coats, lymph is effused between them. In chronic cases, in emaciated subjects, the colon can be grasped through the parietes, as a patient of mine once expressed it, "like a ruler." Sometimes the coats of the intestine themselves become thickened and fleshy-looking.

Cicatrization of Intestinal Ulcers.—The process has been well decribed by Drs. Parkes and Baly. A fibrinous exudation takes place on the floor of the ulcer, which becomes organized. The edges, after being rounded, are drawn down to meet the floor, and from them a delicate lamina shows inwards till the whole surface is covered (Baly).

Exudation.—I have already described an exudative process as being seen at an early period of the disease in the solitary glands, to which, however, it is not confined; it soon fills the neighbouring tubular glands, and spreads over portions of the mucous membrane of the colon and rectum, sometimes, but in India rarely, and then only in chronic cases, extending over the entire surface of the intestine, and even passing into the ileum. Usually of a yellow or grey colour, it forms a thin granular layer: when this is detached, the subjacent membrane is found to be vascular and red. This exudation, when microscopically examined, is composed of epithelium and fibrinous granules, but in the severer forms, Dr. Aitken describes it as consisting of "fine germs with nuclei, mixed with elongated cell-forms," (connective tissue cells).

This exudation is either detached in fragments with much griping and distress, or, becoming to a certain extent organized, it is finally, by a process of ulceration, detached in tubular portions, a process, as shown by Morehead, to be sometimes attended with hæmorrhage.

In Chronic Dysentery.—There is no more common error than to suppose that the symptoms in chronic Dysentery are invariably due to the presence of unhealed ulcers in some portion of the mucous membrane. We constantly see cases in which not a single breach of continuity is found. There is, in such examples, abundant evidence of previous ulceration, and the whole surface of the mucous membrane will be found thickened by a deposition of black granular matter, the result, as Dr. Aitken thinks, "of excessive vascular action and of subsequent changes in the extravasated blood." In other cases abundant ulceration is found, particularly in the rectum and sigmoid flexure of the colon, the ulcers being in every condition, some cicatrized, others undergoing that process, most without vitality enough for healing. Sometimes the whole of the alimentary canal is in a state of atrophy, the grandular structures having disappeared, and the tissues being so attenuated that they are quite transparent. In such cases the general emaciation of the sufferer is extreme. In more recent cases great thickening of the connective tissues and even of the coats of the intestine will be found. In this condition, the exudation is usually found to have undergone the process of organization already mentioned, and it may be found in a state of ulceration like any part of the natural tissue (Aitken).

The coincidence of Dysentery and hepatic abscess is one of the most familiar facts in pathology. But it is often assumed because no abscess is found at a *post mortem* examination in a case of Dysentery, that the liver is normal. Now, in a case of tropical Dysentery it is extremely rare to find the liver healthy. This need not excite surprise, for when to the predisposing, or exciting causes of the disease we add a high temperature, a diet too stimulating for the climate, and habits of intemperance, we have enough to explain the various hepatic diseases, functional and organic, which so often in tropical climates seriously complicate Dysentery. And this without having recourse to any theory of blood poisoning by the absorption of discharges from the sloughing intestine. This view is strengthened by a consideration of the fact that liver abscess is exceedingly rare in the Dysentery of temperate climates. In all Dr. Baly's cases in the Millbank Penitentiary there was not a single case of hepatic abscess.

PROGNOSIS.—The grounds for a favourable prognosis are, (1) The original slightness of the attack. (2) The absence of much nervous depression. (3) A natural countenance, with a pulse of good strength and moderate frequency. (4) The absence of gangrenous odour in the stools. (5) The early appearance of feculence in the stools; and lastly, the absence of any signs of serious hepatic complication.

On the other hand, rapid failure of the nervous and circulatory systems; a pulse increasing in frequency and failing in strength; an anxious and sodden countenance; extreme restlessness; sudden subsiding of pain with increasing fœtor of the stools of a gangrenous character; hæmorrhage from the bowels, mouth, or nose; hiccup; black and dry tongue; suppression of urine, and delirium, all point to extreme danger.

TREATMENT.[1]—It is impossible to over-estimate the importance of early treatment in tropical Dysentery. So rapid is the progress of the disease, so terrible the lesions it causes in a space of time apparently insignificant, that no pains should be spared, more particularly by military and naval surgeons, to impress on the minds of those committed to their charge of the necessity of seeking medical aid on the first manifestation of the symptoms of the disease.

Mild Dysentery.—In this form I have much confidence in the use of the hot bath as a powerful means of restoring the suppressed action of the skin. The bath should be brought to the bedside of the patient and should be maintained at a high temperature, and the patient should be kept in it until he feels faint. After being quickly and carefully dried he should be put to bed, and a dose, not less than from fifteen to twenty grains, of ipecacuanha should be given in the manner to be presently explained. This may require to be repeated in eight or ten hours.

If the patient abstains from all fluid for some hours after taking the medicine, it is seldom that much nausea or vomiting is produced, provided the horizontal position is maintained, which it ought to be.

The result generally is free action of the skin, rapid subsidence of griping, and the appearance of feculent motions. In some cases it may be necessary to give a few drachms of fresh castor-oil, guarded by a few minims of tincture of opium, or a few drops of chloroform. And it is well also to stimulate the abdominal surface by the application of a few turpentine stupes.

The above simple treatment will suffice in a great many cases of that mild form of disease which follows chills without much charging of the system with malaria, provided it be had recourse to sufficiently early.

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[1] In every form of Dysentery both in public and private practice, the stools ought to be received in glazed vessels containing some disinfectant solution, instantly removed from the house or hospital, and carefully and deeply buried. I cannot too earnestly impress this caution on the minds of military and naval surgeons.

Morehead speaks slightingly of the hot bath in the treatment of any form of Dysentery, but I have found it to be a most useful remedy, used with the precautions and in the manner advised above.

Acute Dysentery.—In this form it is even of more importance than before that the patient should be early seen and treated. He should at once be ordered to bed, and as quickly as possible brought under the influence of ipecacuanha in large doses. Some insist on the propriety of first giving a full dose of Battley's sedative, tincture of opium, or a few drops of chloroform, with the intention of making the stomach tolerant of the remedy, and restraining nausea and vomiting.

I believe that the sedative in some cases is useful, and acts in the manner just described. On the other hand, I have often seen ipecacuanha do its work well, and with little disturbance of the stomach, without opium. Should it be determined to premise opium, thirty drops of the tincture should be given, and in half an hour followed by from twenty-five to thirty grains of ipecacuanha, which should be given in as small a quantity of fluid as possible; a little syrup of orange-peel covers the taste as well as anything else. As already advised, the patient should keep perfectly still, and abstain from fluid for at least three hours. If thirsty, he may suck a little ice, or a teaspoonful of cold water at a time may be allowed.

It is seldom that under this management nausea is excessive, and vomiting is rarely troublesome, seldom setting in for at least two hours after the medicine has been taken. The abdomen should be covered with a large sinapism, or a sheet of spongio-piline sprinkled with a little turpentine after being wrung out of hot water.

In from eight to ten hours, according to the urgency of the symptoms, and the effect produced by the first dose, ipecacuanha in a reduced dose should be repeated, with the same precautions as before. All who have had opportunities of trying this mode of treating Dysentery can bear testimony to the surprising effects that often follow the administration of one or two doses of ipecacuanha given in this manner. The tormina and tenesmus subside, the motions quickly become feculent, blood and slime disappear, and often, after profuse action of the skin, the patient falls into a tranquil sleep and awakens refreshed. The treatment may require to be continued for some days, the medicine being given in diminished doses, care being taken to allow a sufficient interval to admit of the patient taking some mild nourishment suited to the stage of the disease.

This, in a few words, is the system of treating acute Dysentery now almost invariably followed in India. It has been long enough in use on a sufficiently large scale to enable us to appeal to statistics and bring it to the test of figures. It has almost entirely superseded the old plan of general and local bleeding, with mercurialisation, either by calomel in scruple doses, or in smaller quantities at short intervals, in combination with opium. Here are the results, derived entirely from official sources:—

Under the old system in Bengal the mortality among Europeans during the forty-two years, from 1812 to 1853-54, amounted to 88·2 in the thousand.

During 1860, when large doses of ipecacuanha were given to the almost complete exclusion of all other methods of cure, the mortality was 28·87 in the thousand.

In the Madras presidency under the old treatment the mortality from the disease during seventeen years was 71 per thousand treated, but when ipecacuanha was largely used, as above described, it fell to 13·5 in the thousand.

In the 44th Regiment, quartered in Fort St. George, Madras, Surgeon Mee treated 68 cases "in the ordinary way"—with a mortality of 6, or 8·8 per cent.

Subsequently 59 were treated with large doses of ipecacuanha and all recovered.

Mr. Docker, surgeon of the 2d Battalion of the 7th Royal Fusiliers, to whom is undoubtedly due the honour of recalling us to a more rational and successful way of treating Dysentery, after he had recourse at the Mauritius to the plan of using large doses of ipecacuanha, lost only one out of fifty-three cases.

I have not space to enter into the complete history of ipecacuanha as a remedy in Dysentery. I must refer my readers to an excellent and exhaustive paper on the subject by Dr. Blacklock of the Madras army, in the Madras Quarterly Journal of Medical Science, and to a paper on the same subject by Dr. Ewart of the Bengal army, in the sixteenth number of the Indian Annals of Medicine. Known from remote times in Peru as a remedy for Dysentery, it was introduced into this country as the *Radix Anti-Dysenterica.* It appears to have been used in Europe in the treatment of the disease more as an adjuvant to other remedies than trusted to alone. In process of time ipecacuanha came to be used almost exclusively as an emetic and expectorant, and, in combination with opium, as a diaphoretic in the form of Dover's powder. In the Madras presidency for many years before the publication of Mr. Docker's paper, it was used as a remedy in Dysentery, chiefly after the example of the late Drs. Geddes and Mortimer, and, as I can testify from twenty-two years' experience of its use, with a marked superiority over the plan of treatment by mercurials. The great merit of Mr. Docker was the introduction of the plan of using it in the large and effective doses of which I have spoken, with the gratifying results given above.

It is probable that ipecacuanha owes much of its usefulness in this disease to its action as an evacuant. It is a blood depurant of an effective kind. It appears to increase the secretion of the whole alimentary canal, as well as that of the liver and pancreas ; under its use tormina and tenesmus disappear, and feculent evacuations are more quickly restored than by any other known remedy.

It also promotes free action of the skin, and exercises a sedative action on the circulation, "In fine," says Dr. Ewart, "ipecacuanha in large doses, may be said to fulfil many indications.

"It produces all the good effects that have been ascribed to blood-letting without robbing the system of one drop of blood, of mercurial and other purgatives without their irritating action, of antimonials and sudorifics without their uncertainty, and of opium without masking the disease." I may add, that it is the most simple, the most successful, the most conservative and the least distressing mode of treatment I have ever seen used in Dysentery. Year by year under its use the number of chronic cases is becoming smaller, and hepatic abscess as a complication is less frequently seen. Although most effective in the early stage of the acute form, it may be given at a much later period with advantage; and even in chronic cases, where from any cause sub-acute symptoms have set in, I often give it with the best results. Some authors caution us against its use in large doses in adynamic cases, and doubtless in such cases very large doses are not advisable. Still, I have used it even where the powers of life were very low, and with the best effect. I well remember the case of a lady, sent to Madras from Calcutta, who landed in such a state of exhaustion that it was with difficulty I could hear her voice. With some misgivings, the symptoms being urgent and the case critical, I gave twenty-grain doses of ipecacuanha at intervals of eight hours, interposing support between the doses; after the third dose this lady was out of danger, and made a rapid recovery.

If unmanageable vomiting follows the use of ipecacuanha, hepatic complication of a serious kind may be suspected—or the vomiting may arise from the system being overcharged with malaria, severe gastric symptoms of this kind being extremely common in remittent fever.

As soon as the disease abates, the dose of the remedy should be abated also. It is well, however, for some days to administer ten or twelve grains at bedtime, for a night or two after the stools are to all appearance healthy. Fomentations, stupes with turpentine, or the application of strong chloroform liniment to the abdomen help to lessen tormina and diminish suffering. If a little diarrhœa, without the dysenteric odour remains, it may be checked with a little astringent mixture, such as the compound chalk powder, with or without opium. Astringents in any shape during the acute stage are not only useless but dangerous.

In Malarious Dysentery quinine in full doses should be given, not less than a scruple in solution some time before the ipecacuanha, and repeated until cinchonism, as evidenced by ringing in the ears, is induced. Ipecacuanha and quinine should be given in alternate doses until the characteristic effects of both are produced. In the malignant Dysentery of camps our utmost efforts must be directed to improve the hygienic conditions of the sick, and in addition to the means already mentioned, carefully modified to suit the condition of our patients, the solution of the pernitrate of iron should be given in full and frequent doses. In some cases I have given ten drops every hour with advantage, and it may be combined with quinine, while the patient is at the same time sustained by milk in small quantities and at short intervals, with beef tea when it can be retained, with wine and brandy when required.

In Scorbutic Dysentery our utmost efforts must be directed to improve the condition of the patient's blood. It is in this form of the disease that fresh Bael fruit has been found so successful in Bengal and other parts of India. I have had occasion to prescribe this frequently in Madras to officers and others who had contracted Scorbutic Dysentery in the province of Pegu, and often with the best effect. Bael fruit has often fallen into disrepute as a remedy in Dysentery, simply from its indiscriminate use. My conviction is that where there is no scorbutic taint it is without efficacy. The Bael fruit is used in India in many forms, as a sherbet, a conserve, a marmalade, or an extract. The former is probably the most efficacious. Sir Ranald Martin in the *Lancet*, and Dr. Alexander Grant of the Bengal Medical Service, in the first volume of the *Indian Annals of Medicine*, have both written interesting accounts of the various modes of preparing and using this remedy. It is doubtless in the same form of the disease that the "Grape Cure" has been found so efficacious. One caution I cannot help giving. I remember the case of a young officer at Secunderabad, who, while convalescing from Acute Dysentery partook freely of grapes. Shortly after, he was seized with all the symptoms of peritonitis from perforation, and rapidly sunk. After death it was found that a grape stone had become entangled in one of the half cicatrized ulcers in the colon, where it acted like a pea-issue; a minute perforation resulted, causing the death of the patient.

Many of the invalids from India, suffering from chronic Dysentery, arrive at Netley in a more or less scorbutic state; all are benefited and some cured, simply by causing them to use whatever fruits are in season.

Chronic Dysentery.—Whenever the disease falls into this stage and resists treatment, the patient should as soon as possible be sent to a better climate. If the locality be malarious, this should be done at once. Often moving him to the sea-coast suffices. More frequently a voyage to Europe is essential to recovery; many lives are lost by delaying this measure until it is too late, and many men are embarked in such an advanced state of disease that they die after being a few days on board. As already stated, the chief mortality among Indian invalids on the voyage home is from Dysentery.

I have elsewhere (Army Medical Reports) insisted on the necessity of extreme care in the management, diet, and clothing of men suffering from the disease at sea. To men in this condition the salt ration is simply destruction, and unless they be warmly clothed on entering high latitudes, they are certain to have their sufferings miserably aggravated.

Whenever the symptoms assume an acute or sub-acute form, the patient ought at once to be placed in bed, and ipecacuanha should be given as recommended in the acute stage, in doses suited to the condition of the patient, and the violence of the symptoms. Gentlemen who have done duty in the clinical wards at Netley have often seen me use this remedy under such circumstances with the happiest effect.

I recommend the use of a water-belt over the abdomen for some hours daily. This acts as a fomentation, and the steady, uniform pressure it maintains seems to favour the absorption of the fibrine effused between the intestinal coats. If there be much uneasiness about the fundament, a water compress over the anus often affords more relief than opiate enemata.

The cold hip-bath should be used daily for a few minutes. I have seen this most serviceable. The greatest attention should be paid to diet. The proper nutrition of the patient is often the most difficult part of the treatment. In those cases where atrophy of the small intestine is much advanced, with perhaps fatty or amyloid degeneration of the liver, or both, it is almost, if not quite, impossible, and the patient dies starved. If the state of the gums point to a scorbutic taint, the diet must be regulated on the principles already laid down under that head. In extreme cases milk must be our chief resource, sometimes with a little lime-water, or beat up with egg, and good sherry or brandy; a teaspoonful of curaçoa added is often highly relished. Rice-flour, sago, arrowroot, or stale bread may be added, and changed so as to suit the capricious appetite. When solid food can be taken without suffering, it should be given; tender mutton should be broiled "quickly and not too much," and eaten with bread and butter. These rules are quite as applicable to the acute stage, where food, while the acute symptoms last, should be simple and farinaceous, and the return to solid food should be gradual.

With regard to medicine, where astringents are indicated, they should clearly be of such a kind as do not tend to lower the already sufficiently low state of the patient. Acetate of lead, sulphate of copper, and suchlike remedies, I use with a sparing hand, and only under the pressure of necessity. My favourite remedy, particularly in men returning from tropical regions, anæmic from loss of blood and the depraving influence of malaria, is the solution of the pernitrate of iron, which I use at Netley very freely, and often with the happiest effect. Under this remedy the whole system often rallies wonderfully, the condition of the blood improves, colour returns to the blanched cheek, the stools becomes more natural and less frequent, the appetite improves, and digestion is more perfectly performed. The citrate of iron and quinine may after a time be substituted.

When astringents of a more direct kind are necessary, the decoction of logwood with lime-water, catechu, gallic acid, and suchlike may be used. Pain must be allayed by opiate enemata, gentle douches to the anus are often most soothing, and the use of liniments containing chloroform, often allay irritation.

In Conclusion.—It will not fail to be remarked that I have not only not advised but by implication have deprecated the use of mercury in all stages and forms of the disease.

My first objection to the use of mercury, particularly in military practice, is one that was forced on me more than twenty-two years ago by observing this fact. Whenever soldiers find that in addition to the misery inseparable from an attack of Dysentery they are to be subjected to that of ptyalism in its mildest degree, they will not present themselves at the hospital gates until further concealment of the complaint is impossible. Secondly, because in ipecacuanha we have a remedy that effects all that mercury can do as an evacuant, without its irritating effects. Thirdly, because experience has shown that men "cured" by mercurial treatment are as a rule cachectic, ex-sanguine, prematurely old-looking, extremely sensitive to atmospheric changes and to relapses from trivial causes. Fourthly, because chronic Dysentery is more frequent after mercurial treatment than when the disease is treated by ipecacuanha. And, lastly, "because men actually under the influence of mercury are very predisposed to the disease" (Morehead.)

In many text-books and works of high authority, blood-letting is still insisted on as an essential part of the treatment of acute Dysentery. I respectfully dissent from this doctrine, first, because, although from the violence of the symptoms there is an appearance of *power*, this is deceptive, for alarming depression often follows free depletion. Secondly, even where the measure appears to relieve symptoms, the heart's action is weakened to such an extent that congestion of the affected mucous membrane remains, a condition which Dr. Blacklock has shown to be nearly as destructive to the tissues as the more acute action. Thirdly, because convalescence after bleeding is tedious. Fourthly, because although bleeding has fallen into disuse, the mortality from Dysentery has decreased. Lastly, it appears to me that the authors who still urge it in this affection and malarial fevers take no account of the state of public opinion on this question. Whatever may be the case elsewhere, a generation has certainly arisen in India that knows not the lancet. Men know, on the other hand, for they see it daily, that those who are not bled recover in a larger proportion than in the days when bleeding was the rule of practice. In short, out of a military hospital where patients have no option but to obey, I assert that it would be simply impossible to carry out the rules for blood-letting which still stand in some of our text-books. I cannot help thinking that treatment of this kind is much in the position of certain penal statutes, which, although still in the statute-book, have become obsolete by the force of enlightened public opinion. I am confident that were it possible even for Robert Jackson, the most sagacious and far-seeing physician the British army ever produced, to reappear once more on the scenes of his former labours, it would be impossible for him, with all the influence of his great name, and all the authority of his vast experience, to induce the men and women of the present day to submit to treatment which they believe to be mischievous, and know to be unnecessary.

DIARRHŒA.

By Edward Goodeve, M.B.

Definition.—Frequent loose alvine evacuations, without tenesmus; due to functional or organic derangement of the small intestines, produced by causes acting locally or constitutionally.

In Diarrhœa the evacuations are liquid or semi-liquid, and vary in quantity, quality, and frequency, with the peculiarities of the disease. They differ in chemical composition, being more or less watery, serous, or mucous, and more or less mixed with the natural contents of the intestines, secretions and other matters poured into the upper part of the alimentary canal. In the acute forms some are dangerous to life, and the same may be said of all the chronic or prolonged forms. The most dangerous of the acute Diarrhœas are those in which there is profuse liquid purging, approaching, in fact, to that of Asiatic cholera.

Nature and Effects.—Diarrhœa cannot be considered as a simple disease. It is rather a symptom of many derangements of health, and is determined by many widely different causes. The matters evacuated come from the small intestines; but though the mucous surface of these is the seat of the flux, the producing causes are often remote from it. Hence these may be separated naturally into two divisions: viz. into such as act locally as irritants, and such as act constitutionally. Both the local and the distant causes may produce either what is called functional disorder, or serious organic disease of the mucous membrane, such as congestion, inflammation, ulceration, thickening and degenerations of tissue. In the official returns, Diarrhœa is classed in the miasmatic order of the zymotic diseases. This may be a proper place for some of the forms; but many do not depend upon any zymotic origin, and ought not, without reservation, to be included in that class.

Diarrhœas are produced differently, according to the nature of the cause. All those which act locally, cause flux by direct irritation of the mucous surface. In all those which depend upon constitutional causes, the fluxes are the means by which the blood is purified of morbid matters; hence they may prove beneficial, as in the instances of critical discharges from the bowels in some fevers and sometimes in gout, &c.; and also, sometimes, by the habitual freeing of the blood from noxious matters, as in albuminuria. In some cases the eliminative process is attended with organic mischief, as in enteric fever, in which deposits and ulcers occur. In some instances probably, not being eliminative, fluxes may be due to reflex disturbances of the functions of the mucous membrane, excited by impressions acting through the nervous system.

It must be remembered that the evacuation depends upon two elements, secretion and muscular contraction, and that these are not always in proportion one to another. The number of stools may vary considerably in twenty-four hours, from three or four to fifteen or twenty; and the frequency may depend upon the quantity of matter secreted, the quality of the secretion, and also upon the degree of irritability of the muscular fibre. In some instances a small quantity of secretion is less easily borne than a larger one in another case, and then the motions will be frequent, though small. In many cases the irritability of the fibre is so great that the contents of the bowels, whatever they may be, are rapidly hurried through the tube, before the lacteals have time to act upon them, so that the degree of irritability of the muscular element is a very important feature in the disease. In some instances the evacuation is caused by reflex muscular action upon taking food or drink into the stomach. In these cases people will say, that immediately after swallowing, their bowels are moved. Young children also frequently have a motion at the time of nursing. Patients think, in these cases, that what they take passes through them immediately. This is obviously not the case.

It has been stated that the evacuations vary in composition. The chief constituents of the stools are mucus, albumen, fibrine, water, and saline matters, mixed with food and natural and morbid secretions. Blood is rare. The chemical nature of the evacuations will not serve as a means of classification of these fluxes. It is very seldom that the same kind of discharge continues throughout the disease. The different products are often mixed together in the same stool, or they vary in quantity from time to time in the same case. It may be conceded that the more fibrinous or mucous the discharges, the more reason there is to suppose that the flux is due to congestive or inflammatory conditions. The stools in Diarrhœa generally differ from those of cholera, in containing more albumen; except, perhaps, in the Diarrhœas produced by hydrogogue purgatives. The consistence and colour of the discharges vary considerably; they may be pultaceous, or as thin as water, or in any intermediate state—the colour is often affected by the proportion of biliary matter, a deficiency of this making them pale; but it also depends upon the colour of the secretions themselves and upon the chemical changes going on within the intestines or in the fæces after voiding. The loose evacuations of young children are often drab or yellow on being passed, and become quite green an hour or two after exposure to the air. When the stools are liquid they separate, on standing, into a fluid and a sedimentary portion; the sediment containing matters, microscopic or otherwise, indicative of mucus, pus, fibrine, and of the debris of food.

The smell of diarrhœal matters may be more or less fæcal; they may be without much smell of any kind; but in some cases they are more or less offensive or fœtid. The chemistry of diarrhœal discharges requires further investigation. It is possible that due research would show that they contain unknown or unsuspected compounds, which would throw light on the diagnosis and the nature of the disease.

Diarrhœa, as will be seen from the above, may be beneficial by freeing the intestines from noxious matters, or by purifying the blood; but if protracted it is always hurtful; the drain of matter from the blood which attends the eliminating act, in the course of time impoverishes it, and all the evils of extreme anæmia come on, and terminate life. This is the case with most chronic Diarrhœas. They may last for months or years and produce exhaustive effects very slowly, but nevertheless progressively. In some of these cases the discharges will probably not be at all copious or frequent, so that the patient's nutrition is not rapidly interfered with. Ultimately, however, the balance between nutrition and drain is lost, and he succumbs. In a certain limited number of persons, Diarrhœa is habitual. Some of these have one or two loose motions daily, for years, without their nutrition suffering much. In these it might be well not to interfere with their condition. They are very rare however.

Diarrhœa in children is readily brought on. It is often very tedious, and frequently dangerous both in the acute and chronic forms.

The influence of Diarrhœa upon life varies greatly in different countries, seasons, and periods, and according to age. The annual mortality of Diarrhœa is greater in hot than in cold climates. In the Indian Sanitary Report it is set down at eighty-six per million in England for seven years, 1848–54, and at 4,555 per million for European troops in Bengal for the fifteen years, 1830–45. In the Indian returns, without doubt, many cases of dysentery are recorded as Diarrhœa. The difficulty of diagnosis is great, and there is much probability that considerable error has arisen from this circumstance; but, after making due allowance for this, there is much greater annual mortality from Diarrhœa in hot than in cold climates, and in the summer seasons of temperate climates than in the winter.

The influence of age is shown in the greater liability of young chidren than adults. The cases and mortality of children under five years of age are much greater than in subsequent ages; but, as Dr. West has shown from the records of the Children's Infirmary, the period of greatest liability is during the period of dentition—from six months to eighteen months or two years of age.

Etiology.—From the difficulty of distinguishing between Diarrhœa and dysentery it may be supposed that the army returns from our foreign stations are not always to be depended upon for showing the proportional prevalence of these diseases; and therefore, in considering the causes of Diarrhœa, it will often have happened that the causes of dysentery have been considerered at the same time. As remarked by

Dr. Ewart (Vital Statistics of the Armies in India), it is probable that it is owing to difference in diagnosis that the relative prevalence of dysentery and Diarrhœa in India appears to differ so much in present and past times. In England there is less room for mistake.

In studying the causes of Diarrhœa we find that a considerable number of them consists of irritating substances introduced into the alimentary canal, but that by far the most important Diarrhœas are expressive of constitutional derangements. Among the causes which act locally, we may place *indigestion, indigestible food, impure food and water, irritating matters or secretions* poured into the bowel, *entozoa.* &c. Those due to constitutional derangements may be secondary to such diseases as *tuberculosis, pyœmia, albuminuria, hepatic diseases,* diseases producing cachexia, as *chronic malarious complaints,* with *enlarged liver and spleen, cancer, scurvy, anœmia, degeneration of tissues of the mucous membrane of the small intestines, disturbances of the functions of other organs giving rise to vicarious fluxes, enteric fevers, gout,* &c. In these the Diarrhœa is symptomatic, but in other instances the causes seem to act more directly on the mucous membrane, giving rise to fluxes without any traceable intermediate diseases, as in the operation of heat, cold, damp, vitiated atmospheres, septic foods and water, and unknown atmospheric influences—some of these probably act through the nervous system and some through the blood.

It is a matter of observation that all cachectic influences have a tendency to produce Diarrhœas ; hence we find that in a very large number of exhausting chronic diseases there is Diarrhœa before death, even although there may be no specific morbid matter to be eliminated, as in the case of enteric fever, tuberculosis, or albuminuria. As the cachexia increases, the blood becomes more and more loaded with effete matter, and the surface of the small intestines becomes the channel of purification by means of Diarrhœa. It may be a matter of doubt how far the cachectic state may be an exciting cause, by requiring matter to be eliminated in the same way as would any inorganic poison introduced into the blood ; or whether this state of the blood merely predisposes the system to Diarrhœa under such exciting causes as local irritants, and the external agencies of cold, damp and vicissitudes of temperature ; certainly any of the ordinary exciting causes of Diarrhœa acting upon individuals in the circumstances above mentioned, will speedily give rise to alvine fluxes. Still, it appears probable that in a certain number of cases the causes act directly as before said, rapidly or slowly causing blood changes. Of course it is very difficult to trace the precise action of the causes in many cases, and without doubt in many we mistake between the predisposing and exciting causes. Diarrhœa often prevails in malarious countries without preceding obvious cachexia, and its mode of production in them affords matter for discussion. It is doubtful whether it arises directly from the action of malaria, or whether this acts merely as a predisposing cause, by which the system becomes readily susceptible to the external impressions causative of Diarrhœa. The high authority of Dr. Morehead is in favour of the view which restricts malaria to a predisposing action only; but with due deference to his opinions, the writer finds it hardly possible to escape the conclusion that in a certain number of cases the flux may come on in an early stage of contamination of the system, before recognisable cachectic influences appear, and where external impressions are not traceable.

In Europe, Diarrhœa, sometimes called sporadic or bilious cholera, or summer Diarrhœa, prevails endemically at certain seasons, especially in the summer or autumn months. It has sometimes been set down to various causes—as heat, heat alternating with cold and damp, checked perspiration and disordered hepatic secretion, and the use of fruits. It is probably due to some atmospheric state; the air being rendered impure by numerous causes in operation, producing miasms, which may be either predisposing or exciting causes in seasons in which the temperature, though high, often varies considerably. Fruit has probably little influence beyond that of an occasional determining cause. Irritative Diarrhœa may be produced by eating too much fruit, but not the genuine severe summer Diarrhœa. Apart from the annual prevalent summer Diarrhœa, in certain years, alvine flux appears to prevail epidemically in England. Thus, in the five years ending with 1848, the deaths in London from Diarrhœa amounted to 7,580, whereas for the five years preceding these they were only 2,828 (Report of Board of Health for 1848-49). This shows that the constitution of the years preceding cholera seems to favour the development of alvine fluxes. A great increase of diarrhœal complaints in like manner preceded the advent of the cholera epidemic of 1832. The mortality from Diarrhœa was unusually large in England during the summer of 1865 (*Lancet*, July 22, 1865). Although we cannot tell what may be the exact causes of these endemic or epidemic fluxes, we find that they are generally much more rife in the hot than in the cold seasons of England, and that the same kinds of atmospheric conditions which favour the spread of Asiatic cholera seem to promote the increase of Diarrhœa. It is true that with high ranges of the thermometer Diarrhœa is nearly always prevalent, but it would be wrong to suppose that heat alone would cause it, neither would great cold. Alternations or sudden changes of temperature may do so, as when the days are hot and the nights cold, especially when there is a predisposition. The Diarrhœas of hot climates are not due to heat alone, nor do they prevail in the hottest and driest part of the year. They depend probably upon several causes combined, such as impure air, malaria, improper food and water. Dr. Ewart's tables show that the European soldier suffers more than the native soldier, while the European officer does not suffer more than the sepoy.

Vitiated and Impure Air.—The influence of this in the production of Diarrhœa may be seen in large standing camps. After a few days' residence, when the conservancy is bad, looseness of the bowels is very prevalent. Medical students and others, who spend many hours daily in the dissecting room, often suffer from Diarrhœa. There are many instances of the production of Diarrhœa when people have been exposed to a concentrated foul atmosphere, by standing close to the mouths of newly opened cesspools, also in those exposed to the emanations of manure manufactories when the wind blew from them towards the sufferers, in those within the reach of the atmosphere of bad graveyards, and indeed to those within the influence of miasms, arising from sewage, fæcal collections, and decaying animal matter, generally. The records of hygienic medicine abound with evidence of these facts.

Indigestible, unusual and impure food, is a very frequent cause of Diarrhœa. The Madras sepoys, stationed on the Tenasserim coast, used to suffer dangerously from chronic Diarrhœa, attributed to changes of diet, and it is well known that the regiments of native infantry, sent from the upper provinces of Hindustan, on their tour of duty in Bengal contract Diarrhœa after a few months' residence in the lower provinces, and suffer considerably therefrom. They exchange the wheaten part of the diet, used by them in Hindustan, for one made up largely of rice, which they find cheaper. In these cases it is not to be supposed that the Diarrhœa is produced by mere irritative causes of unusual food, though this has an influence, but by the gradual deterioration of health, sometimes through scurvy, which requires flux for elimination of morbid matters, or makes the people more readily fall a prey to exciting causes. All large collections of people, as prisoners in jails, &c. suffer from Diarrhœa, and often fatally, when food, water, and air are bad. The evil effects of unadapted diet are shown in the excessive mortality from Diarrhœa in infants and young children who are fed improperly, or brought up by hand, as in some foundling hospitals. It is rare for the child of an Indian wet-nurse to live after its mother has separated from it. Diarrhœa often destroys the infants of wet-nurses in England also. A nurse with bad milk generally induces Diarrhœa in her suckling. The injury of improper diet is also shown by the great mortality from Diarrhœa among the children of the manufacturing populations of the large towns in England. This is well illustrated in Dr. Greenhow's Report to the Privy Council for the year 1861.

Bad Food is often combined with insufficient food, and this last alone, even where the quality of the food is not altered, appears to be sufficient to cause Diarrhœa. This was shown at the Millbank Penitentiary in 1823, when the prisoners had their food reduced from 31 to 33 ozs., to 21 ozs. of solid food daily, with an almost entire exclusion of animal food. The health of the inmates gave way in a few months, Diarrhœa, dysentery, &c. then came on, preceded by signs of deteriorated health (Carpenter's Physiology, 6th Edition, page 57). Diarrhœa frequently attends starvation, and, as remarked by Dr. Carpenter, in the chronic inanition which attends many lingering diseases, the badly nourished tissues furnish the blood with rapidly decomposing materials, and thus cause colliquative Diarrhœas. Bad cooking in large establishments has been known to produce Diarrhœa.

Impure Water.—This is an efficient cause. When water contains much saline matter, it causes looseness of an irritative kind, especially to newcomers into a district supplied with such water. In the process of time they may become accustomed to it and not suffer from it. Water contaminated with decomposing animal matter, or with sewage or sewage gases, is pretty certain to cause Diarrhœa, either quickly or slowly, according to the degree of vitiation of the water and the mode in which it is used. Dr. Parkes mentions that probably 3—10 grains per gallon of putrescent animal matter may suffice to render water hurtful. When Diarrhœa prevails over a limited district, or in a few houses, or in a single house, the water will very frequently be found to be the cause, and the supply should be submitted to close examination. Water rendered impure by animal matter may cause Diarrhœa by direct irritation, but more probably by the septic matters absorbed into the blood. The Hill Diarrhœas of some of our Himalayan stations have been attributed to the water, but Dr. A. Grant thinks that though this may have some influence, it is only in combination with impure air and malaria.

Bad dwellings, or habitations which are damp, cold, dark and unventilated, and which are too near the ground and badly drained; and *insufficient clothing*, should all be considered as causes of Diarrhœa.

The seasons in which Diarrhœa prevails most in Europe, are in the summer months; from June to September, or earlier in the year if the temperature is unusually high. The same law operates in the case of children, as was observed during eight years in the London Children's Infirmary (West, Diseases of Infancy and Childhood). In India, Diarrhœas prevail in the rainy season, and not seriously in the hot and dry months of the year. Bowel complaints are very frequent, however, in the cold season in Bengal and in Bombay. This applies both to natives and Europeans. It is very probable, as supposed by Dr. Morehead, that in the cases occurring in the cold season, the cold and damp weather act as exciting causes upon cachectic persons.

In summing up the causes of Diarrhœa, we find them to be—

1. Such as produce direct irritation of the bowels.
2. Such as, by rendering the blood impure through primary diseases, produce Diarrhœa as an eliminating flux; or render the body more susceptible to the action of exciting causes.
3. Such as act directly on the system through cold, heat, impure air, malaria, crowding, bad or insufficient food, or water; and which determine an eliminating Diarrhœa; or act, as in the case of vicissitudes of temperature, through impressions upon the nervous system only.

FORMS AND SYMPTOMS.—We need not consider more than the following forms.

1. Irritative Diarrhœa.

2. Congestive and Inflammatory Diarrhœa.
3. Summer Diarrhœa.
4. Chronic Diarrhœa.

1. *Irritative Diarrhœa.*—This depends upon the action of matters which have in some manner entered the intestines. The common feculent Diarrhœa is an example of its milder form.

The irritating matters may be indigestible or undigested food, badly cooked or diseased or putrid food, shell-fish, mushrooms, fruits and vegetables, diseased secretions from the intestinal walls, and secretions from ulcers in any part of the alimentary canal above the lower end of the ileum, irritating matters poured into the bowel, as in the case of vitiated biliary secretions, abscesses opening through the intestinal walls, entozoa, purgative medicines, and irritant poisons. These have various degrees of effect.

Common feculent Diarrhœa.—In this, a few hours after a meal, the patient feels colic and flatulence, and a desire to evacuate the bowels. There is often nausea and foul tongue; seldom vomiting. The pain is often relieved by the purging which ensues. The stools have a feculent character, and are of brown fluid containing lumps of fæces, often offensive. The colour becomes lighter after four or five evacuations, and if the disease goes on may end with mucous stools, but generally the looseness passes away after a few motions which have removed offending matters. The pulse and the strength do not change, or but slightly. The milder irritants may excite this form of purging. It is sometimes caused by over eating, or eating too rapidly, independently of improper articles of food, or by temporary states of indigestion due to fatigue, or other disturbances of digestion which interfere with the proper chymification of the usual diet. It may sometimes be prolonged by continuance in excess of food. In some plethoric persons, Diarrhœas of this sort seem to be excited by critical irritating discharges from the intestines themselves.

Much more severe symptoms attend when the irritating matters are diseased or putrid food or water. The Diarrhœa is more violent and exhaustive, the constitutional depression very decided, as shown in the feeble pulse and coldness of surface. The stools may be offensive at first, and attended with much flatulence, and the disease may continue after the offending matters are expelled, owing to induced inflammation of the mucous membrane, which may cause organic changes. The symptoms of this form are probably not always due to purging only, but also to the septic influences of the decayed ingesta. These cases resemble the milder forms of cholera in the violence of their symptoms. They generally recover in a day or two.

Lienteric Diarrhœa.—In this form of irritative flux there is, with the frequency of evacuation, a want of assimilation of food, which passes through the intestines more or less unaltered, and causes irritation in its passage. This is usually associated with the chronic forms of the disease, and may complicate all of them. The Diarrhœa is not always due to the state of the ingesta only, but often exists as a separate complaint; and indeed often causes the imperfect assimilation by its debilitating influence upon the general system, and on the process of digestion; in these cases the disorders of the stomach and of the bowels act and react injuriously on each other. In lientery the stools are frequent, mucous or serous, more or less coloured with bile, or of buff colour, or straw colour, or greyish, and mixed with undigested food. In young children this is frequently seen both in acute and chronic Diarrhœa, the stools being composed of unchanged casein. In some cases the food is partially assimilated, or some kinds of food are acted on while others are not at all changed. In lientery the patient emaciates rapidly, although he may have a good and even large appetite. The drain and the absence of nutrition both contribute to reduce him. Even that portion of the food which may be digested is hurried onwards, owing to the irritable state of the muscular fibre, so that little or no absorption can take place, and thus the supply of nutriment available to the system is reduced to a minimum.

Bilious Diarrhœa.—This may be acute. Excessive biliary secretion is said to be the irritating cause. The patient passes frequent bilious motions, has scalding sensations at the anus, and griping pains. The motions are frequent, green or yellow, and if the disease continue three or four days, like other varieties of irritative Diarrhœa, it is liable to pass into the congestive or inflammatory forms, and the motions may contain much mucus.

This form of Diarrhœa, due to the direct irritation of biliary secretion, is perhaps not so common as is supposed by some writers. In hot climates it is probable that the so-called bilious Diarrhœa is really dysentery rather than an affection of the small intestines. Even in this, however, the secretion of bile in excess is rather secondary to intestinal irritation than a cause of it. The flow of bile is readily excited by reflex action when the intestines are irritated. Still, without doubt, Diarrhœa may be caused by excessive biliary secretion, and sometimes this does exist as an irritating cause, and does produce the effects just mentioned.

In climates in which liver diseases are frequent it is always necessary to look to the condition of the liver in cases of Diarrhœa which are at all protracted. It is probable that Diarrhœas, due to vitiated bile, are not nearly so frequent as is supposed, but still we often find deranged biliary functions with Diarrhœa, and should always remember the connexion and investigate the state of the liver in all cases of intestinal flux. Diarrhœa sometimes exists with suppuration in the liver, but it is doubtful whether any particular form of the flux is so associated. Dr. Parkes thinks that copious, yeasty, light-coloured stools are often met with in liver abscess. Annesley mentions dark-coloured stools. It is probable that no particular form of flux is diagnostic, but whenever we find a continuance of the stools before described, we should always be alive to the possibility of hepatic disease.

Entozoa.—Diarrhœa does not necessarily occur with these, but they give rise to it occasionally, especially in India, and should always be thought of as a cause in obstinate and obscure cases. It is more particularly active in children. The lumbrici apparently irritate the bowels more than the tape-worm, but this last does so occasionally. It may be that this appears so, simply because the lumbricus is more common than the tape-worm. In these cases the stools contain mucus. This is not always the case, but when there are mucous stools without the symptoms which generally attend the sub-inflammatory forms of Diarrhœa, we should do well to suspect the presence of entozoa. Trichinæ cause Diarrhœa, but other symptoms ensue which soon throw the Diarrhœa into the shade.

It is clinically important that we should recognise Diarrhœa from the irritation of *morbid substances poured into the intestines,* as from abscesses or other fluid collections opening into it. This is more frequently seen in India than in Europe. In India an occasional cause of Diarrhœa is the opening of hepatic abscesses into the small intestines. A sudden and perhaps profuse evacuation, preceded perhaps also by some relaxation of the bowels, should cause suspicion of the opening of a large liver abscess, but the particular diagnosis of this refers rather to that of hepatic disease than to Diarrhœa.

Any of the irritative forms of Diarrhœa may pass on to chronic Diarrhœa by exciting permanent disease of the intestines; or may be prolonged owing to a continuation of the exciting causes, as in the cases of bad diet, bad water, &c. and which would cease on removal of such sources of irritation.

2. *Congestive or Inflammatory Diarrhœa. The Mucous and Serous Diarrhœas of some writers.*—Any of the forms of Diarrhœa before-mentioned may give rise to organic disease of the mucous membrane by originating congestions or inflammations, ulceration, &c.; but these changes may also arise from constitutional causes, such as exposure to cold, checked perspiration, miasmatic influences, independently of local irritations, and be the sources of the serous, mucous and fibrinous discharges which characterise these forms. The milder forms of inflammation are classed under Diarrhœa, though they would be more properly placed under the head of sub-acute or chronic muco-enteritis, but as the flux is the principal symptom, they are often described under Diarrhœa. Anatomically, the parts involved are the mucous membranes, glands and follicles. The stools in this vary considerably in character. In the early stages they may be liquid or albuminous, or be a mixture of mucous and watery fluids; sometimes they are called serous. At times they contain glairy and opaque or stringy mucus, or consist mainly of mucus with some fæcal matter, and occasionally fibrinous or flaky masses. The stools may be frequent—three or four to ten or twelve in twenty-four hours. They are rarely offensive. They may be copious in the watery forms, but are often not large when they are chiefly mucous. There is more or less constitutional disturbance, slight quickening of the pulse, with slight or no febrile reaction. The abdomen feels sore on pressure, and is

sometimes somewhat distended. The tongue may be reddish, but at other times not altered. The patient often complains of griping, gnawing, or pricking pains. There is loss of appetite, or nausea. In a few days the disease generally yields, if not, the patient emaciates; so, if the disease passes into a chronic state, the skin ultimately becomes harsh and dry, and as the complaint proceeds there is often great exhaustion, and it may be fatal with the usual symptoms of chronic Diarrhœa. In young children it is often fatal in the acute stages; the evacuations are frequent, copious, thin, glairy, perhaps tinged yellow. In many cases there is much straining, and blood appears in the stools. These last are cases of dysentery rather than Diarrhœa, but in the beginning there is extreme difficulty in knowing what the disease is to be, and indeed the diseases are much mixed up together. In the diarrhœal form exhaustion soon comes on if the disease is not checked, and death may take place in three or four days. This is called "watery gripes." It may pass into a chronic state in the child as well as in the adult. In some instances of the inflammatory Diarrhœas, ulceration may be diagnosed in the protracted cases by small masses or striæ of blood being found in the discharges. They would probably be more frequently found if the sediments of the stools were more commonly submitted to microscopic examination. In typhoid fever ulceration sometimes gives rise to decided hæmorrhage. In some of the more chronic forms of these Diarrhœas, the discharges are quite pale, and some writers supposing that this is due to excessive secretion from the mucous follicles, have called it *Chylous Diarrhœa.* It is probable that in these cases there is imperfect or deficient secretion of the bile, as well as unabsorbed chylous matter (Copland's Dictionary). In some cases there is fibrinous exudation, which takes the shape of the surface from which it is exuded, and thus complete cylinders or casts of portions of the small intestines may be formed. These are sometimes several inches long, are of considerable consistence, and may be thicker than the coats of the bowel itself. Dr. M. Good has called the disease *Diarrhœa tubularis* when the exudation is thrown off and appears in the stools in a cylindrical form. More frequently, membranous patches are detached, and occasionally long standing looseness, griping, &c. cease immediately after the expulsion of a piece or two of this membrane, which while attached to the surface of the mucous membrane had probably caused the symptoms.

These inflammatory conditions of the mucous membrane may, as before said, be caused by persistent local irritation, or by any of the primary diseases, or by the various external agents before mentioned. The disease may yield in the early stages, but on the other hand the most grave consequences, such as thickening, ulceration and contraction may ensue, and the Diarrhœa become chronic. In the nature of the discharges these forms of Diarrhœa so nearly resemble those of dysentery that it is often impossible to distinguish one from the other, especially in the chronic stages, and indeed it seems that in young children both the small intestines and the colon are often simultaneously involved.

3. *Summer Diarrhœa. Sporadic Cholera or Bilious Cholera.*—This form is generally met with in the north of Europe in the summer months, from June till the end of September.

The disease generally sets in suddenly. There is vomiting and purging. The vomiting is copious. The stools are also copious and watery, and often expelled with force; they are generally loaded with bile, and of dark brown or green colour, as are the vomited matters after the stomach has been cleared of food. There is much painful contraction or cramp of the muscles of the abdomen, of the muscles of the extremities and of the bowels, recurring frequently. The tongue is dryish and there is much thirst. Exhaustion comes on rapidly in this disease, so that in a few hours the pulse, though firm and even full at first, will have sunk, the voice have become feeble, the skin bedewed with cold or clammy perspiration, the temperature of the body reduced, and a sense of sinking, and faintness will be established. In severe cases symptoms of decided collapse succeed, and the patient may die. The disease is not so rapid as Asiatic cholera, and the mortality is small. People seldom die under two or three days of illness, but in extreme cases it has been fatal within twenty-four hours. The urine is seldom quite suppressed. In some cases gastro-enteric irritation or some febrile disturbance follows the attack. As far as symptoms go the disease is not so violent as Asiatic cholera, but they seem to differ rather in degree than in kind.

Although the relation of this disease to Asiatic cholera has not been made out, it is impossible to avoid speculating on the resemblance between them. The presence of bile in the stools, the greater griping, the longer duration of the disease, the non-suppression of urine, and the smaller fatality, seem to be the only points which distinguish summer Diarrhœa from epidemic cholera. These differences are not so obvious in all cases, and in some of the worst forms of the English disease the complaint would at once be recognised as real cholera if it occurred during an epidemic of Asiatic cholera. True choleraic Diarrhœa often continues for days with bile in the stools, and may ultimately pass into pure cholera. Mild Diarrhœas of bilious character are frequently met with in the summer months in Europe. They last a few days, and are generally amenable to treatment. It will not be necessary to say more about them.

4. *Chronic Diarrhœa.—White Flux.—Cachectic Diarrhœa.*—It will be sufficient to speak of the chronic forms of all the foregoing varieties of Diarrhœa in one section, and in this also to include the insidious forms of Diarrhœa which begin without acute symptoms, and which, from the paleness of the stools, are sometimes called *White Flux.* The tendency of all chronic Diarrhœa is to destroy life by anæmia and exhaustion, so that in describing the so-called white flux the progress of the others will be illustrated.

Chronic Diarrhœa, beginning insidiously, is not uncommon in India, nor probably in other parts of the world. It attacks Europeans and natives, and is not confined to people in poor circumstances. There is no protection in length of residence; indeed, the old European residents suffer more than new arrivals. Deterioration of health from climate favours the disease known as white flux. Though called white, the evacuations are not often very white. They are very pale, however, especially after they have lasted any time. This flux probably arises independently of local irritation, though it may be aggravated by such. It has a dangerous tendency when firmly established in the system, though it may take months or years to run its course. At times it progresses more rapidly. It is often met with in people who have in some way suffered from malaria, but it also attacks those who do not show decided signs of it. A somewhat similar disease prevails in the Indian Hill stations of Simla, Kussowli, and Dugshaie, but those of Darjeeling, Nynee Ial, and Mussourie, seem to be free from it. It is probably due to the direct or indirect action of malaria acting feebly and slowly on the system.

This dangerous disease begins without marked symptoms beyond those of relaxed bowels. There may in some cases, even at first, be only two or three stools in the twenty-four hours, and the patient may pay little attention to them. They may be more frequent than this, however, and may vary from slightly loose and pultaceous motions to copious and fluid ones. They are generally yellow, light yellow, or pale drab or whitish in colour. When liquid, there is not much sediment, and little or no mucus; they are often passed with force. As the disease advances the paleness of the evacuations is more constant, and sometimes they have the appearance of chalk and dirty water, are very liquid and frothy; sometimes they are of the consistence of thickish gruel. There are generally several evacuations in the twenty-four hours. In one case I saw but one motion in twenty-four hours, but that was large and expelled with force. In no single case are the evacuations always of the same character. They frequently vary within certain limits; but it may be said that in the established disease they are more or less pale or approaching to whiteness, and more or less liquid, becoming lienteric, and sometimes dysenteric, in the later stages, if the colon becomes implicated, as may happen. The cases vary in their associated symptoms. In some, dyspeptic symptoms are very troublesome. The tongue is sore, smooth, and shining, and perhaps studded with small aphthous ulcers, which may also attack the lips. The patient complains bitterly of the feeling of soreness in the mouth, and of the pain in eating or drinking. The abdomen is often distended with gas, and distressing flatulence and colic follow every meal. The appetite is often considerable, very capricious, and there is a craving for unsuitable food, which is often indulged in, in spite of the most serious warnings. In other cases the dyspeptic signs are not severe, the tongue is pale and soft, the abdomen flat or but little distended. Although in some cases the appetite is great, in the majority of cases it is lost or indifferent. In general there is not much or any pain; the characteristic feature being that of painless, emaciating, exhausting Diarrhœa. In the advanced stages the temper becomes irritable, the pulse thready but not always quick, sometimes indeed slow; the skin generally harsh and dry, parchment-like, or in others soft and silky, and very often brownish or darkish in hue, or the tint of the brownish skins deepened. With all this, emaciation progresses as does failure of bodily strength. Great anæmia follows, as shown by the pearly eye and bloodless conjunctiva, the pallor and the dropsical swellings. Articular pains without swelling, and scorbutic patches, occasionally appear, and in the natives of India ulcerations of the cornea. The patients die with more or less anasarca, creamy exudations on the tongue and inside of mouth; sometimes with coma or convulsion, and occasionally from syncope during slight exertion. When recovery takes place there is great risk of relapse under exposure or other exciting causes.

It is difficult to tell the exact mortality of this disease. It is considerable in the confirmed cases.

The paleness of the discharges has obtained for this disease the name of white flux, though probably the term Diarrhœa alba has been differently applied by different authors. This paleness has led many to think that the exciting cause of the disease is the state of the liver secretion. It is probable that too much importance has been attached to this as a cause of the flux, and that the liver derangement is merely a part of the great general disease which gave rise to blood changes. Without doubt, an absence of bile in the upper part of the canal interferes greatly with the digestive processes, and thus the Diarrhœa may be aggravated in the same way as it is when the stomach digestion is imperfect, and therefore it is of importance that the biliary secretion should be restored. We must remember, however, that in judging of the action of the liver the colouring matter is not the certain measure of the quantity of biliary acids secreted, and we should, in estimating the quantity of colouring matter, recollect the quantity of liquid and the colour of the secretions with which it may be diluted or mixed. There can be little doubt, however, that there is a deficiency of the colouring matter of the bile. It may be noticed that in all forms of chronic Diarrhœa there is a tendency to paleness of stools as the disease advances.

It is not improbable that there may be more than one form of chronic constitutional Diarrhœa in India and elsewhere, but in the majority of cases it will be found that they are secondary to scurvy, recognisable malarious disease, &c.

Hill Diarrhœa.—Under the name of this, and of Diarrhœa alba, passing into Diarrhœa cum cachexia, or cum anæmia, Surgeon Major Alexander Grant has described the disease of the Indian Hill stations, which resembles in so many particulars the white flux of the plains. This valuable paper is to be found in the first volume of the "Indian Annals," and should be carefully perused.

Dr. Grant thinks that there is more decided derangement of the functions of the liver in the hill disease than in that of the plains. The stools are pale drab or muddy grey colour, frothy, and feculent in the early stages; but they are often very white. In later periods they become yeasty, pultaceous, or lienteric. The stools are generally passed between daylight and breakfast time, and again as evening approaches. Their odour is peculiar and unnatural, but not offensive. The digestive organs are disturbed in the same way as they are in the plains. If the disease advances unchecked, anæmia and exhaustion occur. It may last for years, but is generally relieved in the early stage by medicines or by change of residence to the plains; but it may require change to Europe. It is dangerous when very chronic or confirmed, and shows a strong tendency to relapse. This relapse often occurs after removal to the plains. Dr. Grant notices this as having been the case in regiments moved from the hills to the low lands. The surgeons who were with the army of the Punjab in 1848–49 will remember how severely the men of H.M. 29th, the 2d Europeans, and the Bengal Artillery suffered in the plains, after having, previous to joining the force, been subject to the disease in the hills.

Dr. Grant attributes the disease to ordinary external causes, but thinks that they must be conjoined with malaria. He says that the disease begins about the time that the rains set in, "At the time when the air, from being dry and warm, becomes moist and cool, and from the hot steaming ravines and valleys heavy vapours ascend and envelop the stations. The nights are cold as well as damp, and exposure to night air causes hepatic congestion and functional derangement."

MORBID ANATOMY.—The morbid changes are various. In the *acute* form little or no structural changes may be found, except perhaps some patches of congestion or slight vascularity. In the cases of young children dying of the inflammatory forms there may be some swelling of the follicles and of Peyer's patches. In the *chronic* forms there may not be much surface change except in the inflammatory varieties. There may be traces of redness in patches, ulceration of the glandular structures, with the floors in various stages of repair, greyness of portions of the mucous membrane, black or dark margins to the ulcerated tissues or healed ulcers, puckering and contraction. In some chronic Diarrhœas, especially in those with white flux, in spite of the long duration of the disease, little or no change of the mucous surface is to be found. There is a great thinning of all the coats of the small intestines, so that they are quite translucent. There is doubtless atrophy of the glandular tissues. These remarks also apply to the state of the intestines in Hill Diarrhœa. When these patients have had dysenteric symptoms towards the close of life, ulceration or sloughing of portions of the colon may be found in them. The mesenteric glands are generally enlarged and hardened in all chronic

Diarrhœas, and especially in the white flux. The liver is not necessarily altered even in the Diarrhœa with white flux. It has been found flaccid and anæmic and small, or of natural size, and without any decided morbid appearance. It occasionally presents patches of fatty degeneration, but this is not peculiar to chronic Diarrhœa.

In some cases of chronic Diarrhœa, some tissues of the small intestines are found in a state of amyloid degeneration. Dr. Aitken, in post-mortem examinations of invalids at Fort Pitt and Netley, has often seen this degeneration involving entire villi, points of villi, mucous and submucous capillaries, and the vessels surrounding the sacculi of Peyer's patches, also in a state of annular infiltration round solitary glands. In these cases it was probably associated with similar degenerations of other organs. It would be well to study carefully the condition of the tissues of the small intestines as to degeneration in the Diarrhœa with white flux, and indeed in all the chronic forms of the disease.

DIAGNOSIS.—Due attention to the history and symptoms will generally enable us to distinguish between the different forms of Diarrhœa. There may be difficulty at the onset in deciding between Diarrhœa and cholera, when the latter disease is epidemic. Information on this point will be found in the article on Cholera.

Some of the forms of dysentery are frequently mistaken for Diarrhœa. In these cases of dysentery the motions are copious, liquid, deep brown, or brownish yellow, and there is straining and griping. These may pass on to a fatal termination without any masses of mucus or blood being discovered by the ordinary way of inspecting the evacuations, and hence the error. It is chiefly in the beginning of these cases that the error occurs, and between them and bilious Diarrhœa. One may feel pretty sure, however, that in tropical climates, in cases in which there are frequent liquid evacuations, with some griping and straining, these are dysenteries, and of a dangerous character. In them there will probably be found some soreness and fulness on careful pressure over the cæcum and sigmoid flexure, which will not be present in Diarrhœa, but this is not always to be made out satisfactorily. In dysentery, by washing the evacuations so as to get rid of all the colouring and fæcal matter, we shall find at the bottom of the vessel more or less reddish or bile-stained mucus in flocculent masses or flakes or strings. This will scarcely ever be found in Diarrhœas, except, perhaps, in those dependent on recognised fevers. Otherwise, mucous Diarrhœas are rare in adults in India.

In deciding between chronic Diarrhœa and chronic dysentery we should always study the history, and we shall probably find that those cases in which there has been an absence of mucus and straining in the early stage are Diarrhœas. In chronic dysentery the mucus is sometimes thin and glairy, and not easily recognised in the evacuations. Bloody evacuations are more frequent in chronic dysentery than in Diarrhœa, but occasionally in these last, with ulceration of the follicles or membranes, blood may be present. In these cases it would be difficult to decide as to the source. In chronic Diarrhœa the quantity is generally much less than in chronic dysentery; in both, however, it may be in mere striæ or minute clots, or diffused in the fluids, and not recognisable except with the microscope.

In England, when the motions contain much mucus, and there are colicky pains, we may conclude that the flux depends upon irritation, congestion, or inflammation of the mucous membrane or follicles.

It is well to remember the possibility of both acute and chronic Diarrhœas being caused by irritant poisons administered either accidentally or purposely.

PROGNOSIS is favourable in the feculent and in the irritative forms not dependent on matters poured in from abscesses, &c. Moderately favourable in the earlier stages of congestive and subacute inflammatory Diarrhœas in adults. Unfavourable in chronic Diarrhœas, and especially so when debility or anæmia or emaciation have begun. Very unfavourable when complicated with disease of liver or spleen, when there is much leucocythæmia, emaciation, scurvy, anasarca, or dropsy. The same may be said of lienteric cases. All these states are still more to be dreaded when circumstances render a change of climate impossible for tropical sufferers. In the last stages of chronic Diarrhœa it often happens that the motions diminish or cease for a few days before death. We should be careful not to give a favourable prognosis in these cases. The cessation is a sign of fatal debility.

TREATMENT.—As Diarrhœa may depend on so many causes, it is highly necessary before treating any particular case to ascertain as far as possible its nature and cause. In many cases it may be desirable to favour, or certainly not to check the fluxes, especially when, as in vicarious or other discharges, they eliminate noxious matters from the blood. In some of the irritative forms of Diarrhœa it may be desirable to assist nature in the expulsion of the offending cause; but in no instance should we consider that the removal of this supposed offending matter is the one and only thing to be aimed at. The general condition of the patient is also to be taken into account, and the effect which the purging may have had upon his system. It may be that the flux has so depressed the powers of life that they must be supported, and that the offending cause must be allowed to take its own course. The determination of this is a matter for pure experience, and must be left to the skill of the practitioner, and to his power of deciding between real and apparent debility. It would be well that no preconceived notions should guide us. In entering upon the treatment of any individual case we should always direct our attention to its particular cause, and consider the food, the water, the cooking, the cooking vessels, the season, and prevailing epidemic influences. In many cases the simple regulation or change of diet will render medicines unnecessary; or the removal of patients from districts in which the water or air is impure, or the supplying them with good and sufficient food and water, will be all that is required. In these cases it will generally be found that more than one person is affected; but this will not always be so, and the affected person may be a new comer, and not able to tolerate influences which have no effect on those habituated to them.

The common *feculent Diarrhœa* generally speedily cures itself by the ejection of the acrid or offending matters, and after four or five motions the patient is relieved and pain ceases. In these cases the patient may be left to himself. In places where no cholera prevails we may, if there be much colic, give a drachm of Gregory's powder (compound rhubarb powder), and a scruple of carbonate of soda, with ten or twenty minims of laudanum, in peppermint or plain water. The laudanum quiets the spasm and assists the action of the aperient. If the looseness continues after this has operated, preparations of chalk and soda, with or without opium, may be given every four or six hours. At bedtime a grain each of opium, calomel, and ipecacuanha may be prescribed for two or three nights. The diet should be light and small in quantity; for the first day little or nothing. The continued administration of aperients in Diarrhœa when the stools become pale or contain mucus is hurtful. When administered in persistent Diarrhœa they should be given rarely. When necessary, the patient may take a grain each of opium, ipecacuanha, and blue pill, at bedtime, and a mild dose of castor-oil in aromatic water in the morning. When the oil has produced two or three stools, the astringent remedies should be resumed. The patient is often debilitated even by such a mild course of purgation as this, and when such is observed, aperients should be avoided. If cholera prevails, opiates should invariably be used without aperients or without regard to supposed offending causes. In the more severe forms of flux, dependent upon diseased or putrid food, &c. the bowels should be evacuated if the strength permits, or if we think that the irritating and depressing substances have not already been voided; but in severe cases we must resort at once to stimulants, aromatics, ammonia, &c., warmth to the extremities, and bags with hot bran to the abdomen. Æther with a little laudanum, and warm brandy-and-water with spices, are often required.

In *summer Diarrhœa*, if the attack approaches choleraic Diarrhœa or cholera itself in character, the means mentioned in the article on cholera should be used—brandy, laudanum, camphor, sinapisms, and the recumbent posture, &c.

In Diarrhœas due to *congestion and inflammation* we should use warm fomentations to the abdomen, counter-irritants or rubefacients, as turpentine epithems and sinapisms, or even blisters, warm baths, with attention to diet and clothing. For internal medicines, Dover's powder with grey powder and soda, Dover's powder with bismuth and soda. Occasional purgatives may be required for this purpose; castor oil in emulsion with a few drops of laudanum will be the best. A dose of blue pill and ipecacuanha may be given at bedtime of the evening before the oil is to be taken. Astringents seldom or never act satisfactorily in the early stages of these forms. Counter-irritation, attention to the secretion of the skin and liver, with opiates and light digestible diet, are most useful. In the later stages astringents may be used with advantage. When entozoa are suspected, vermicide medicines with aperients may be tried.

In the *chronic forms* of Diarrhœa of all kinds the treatment is often very unsatisfactory. They often resist all remedies however carefully or judiciously they may be prescribed. This disease requires the full co-operation of the patient in all its stages. No special treatment is required for any particular form, so that the following observations, which chiefly apply to the white fluxes, will answer for analogous stages of any of the chronic forms. It is desirable that the diarrhœal drain should be stopped or reduced, but this will not be done by astringents only, without the improvement of the general health. We should endeavour to restore the function of the liver and skin, if impaired. We may assist the action of the liver by ipecacuanha, taraxacum, and nitric and muriatic acids. We must not suppose that these will be useful unless the patient's general health improves. We cannot expect the liver to furnish much biliary matter under special stimulants in the anæmic conditions of patients in the advanced stages of Diarrhœa. When the health mends, the secretions will improve also. The remedies mentioned above will not injure even under prolonged use, but mercury should not be given in these cases. Sir R. Martin has found nitric and muriatic acid used internally and externally very efficacious in these cases, and they should be tried. I suspect that these remedies are more useful in Diarrhœas with enlarged livers than where there is anæmia and deficient bile secretion without organic change in the liver. Twining recommended laudanum and nitric acid, and gave a case of rapid cure thereby. It may be given in doses of 10 to 15 minims of dilute nitric acid, and 15 to 20 minims of laudanum in calumba or gentian infusion or plain water, three times a day. In the anæmic stages I have sometimes seen benefit from 5 to 10-grain doses of the potassio-tartrate of iron, with 10 to 20 of laudanum in cinnamon water three times a day; also from the simple tincture of the muriate of iron. The iron alum is also a good preparation in 3 to 5-grain doses. Preparations of strychnine, with iron and opium, or the extract of nux vomica, or the powder of the seed, should always be tried; they appear to impart tone to the stomach and bowels, and are very valuable in some cases.

It would be well to use quinine in all cases in which malaria may be supposed to be operative. 5 to 10 grains may be given daily in divided doses, with an excess of sulphuric acid and with laudanum. If this irritates the bowels, it might be tried in the form of pill with opium, or in enemata with laudanum. Dr. Aitken mentions that salicine in 5-grain doses three times a day has been found very valuable in bad cases with a clean tongue, in which opiates and astringents had entirely failed. Bitter tonic medicines sometimes assist the digestion and improve the appetite, and opportunities should be taken of administering them in cases in which the stomach will bear them; they should be such as can be combined with the other remedies, such as iron, strychnine, opium, &c. The natives of India have for a long time used the Bael fruit in prolonged Diarrhœa. It seems to give tone to the bowels and to regulate them. In India, when the fresh fruit can be obtained, the natives roast it slightly and eat the inside pulp; one or two fruits daily. The Europeans generally use it in the form of a sherbet of creamy consistence, which may be made by scooping out the inside of the ripe or partially ripe fruit, mixing it with a little water to the consistence of pap, and removing all the stringy parts with a fork. A small tumblerful of this mixed with sugar is generally taken in the morning before breakfast. I have sometimes seen benefit from its use, but have been frequently much disappointed in its effects. Dr. A. Grant mentions that he has often found it fail in the treatment of the Hill Diarrhœa. Bael disagrees when the digestion is feeble, producing flatulency and acidity, and spoiling the appetite. In such cases it might be well to take it at bedtime instead of in the morning. In Calcutta two or three preparations of the fruit are kept at the druggists', and may be used when the fresh fruit cannot be obtained. Some are also procurable in England.

In the treatment of Hill Diarrhœa, Dr. Grant observes that early and prompt cures follow treatment in the early stages. The great object is to restore the function of the liver and skin. This may be effected by at first giving a dose of blue pill and Dover's powder at bedtime, and castor oil with a little laudanum in the morning; repeating the remedies if necessary, and keeping up an astringent action on the bowels by occasional doses of hydrargyrum cum cretâ and Dover's powder, or by the compound powder of chalk, with one-fourth or one-half of a grain of morphia. He advises that opium should not be given except with ipecacuanha. In obstinate cases he trusts almost entirely to a combination of aromatics and astringents, concurrent with a strict attention to diet and clothing. He strongly objects to the use of mercury in the chronic stages, and urges change of climate. He has found benefit from the use of bromide of potassium in cases in which he has suspected the enlargement of the mesenteric glands.

It may be well to say a few words about the use of opiates and astringents, remedies which are so much used in Diarrhœa. Both are very valuable, but require discrimination in their use. *Opium.*—This is used largely by the natives of India for Diarrhœa, and many confirmed opium-eaters have acquired the habit by taking the drug for alvine fluxes. I confess that I have not seen it attended with the evils which some writers have. I think that the bad effect upon the secretions is much exaggerated. In health the hepatic secretion is certainly temporarily diminished or suspended by its use; but is this always its effect in disease? After much observation during the use of opium I think that it is not. But at any rate it does not injuriously influence the liver secretion when combined with ipecacuanha. On the skin the combination acts freely. Opium seems to act less as an astringent than as a sedative to the peristaltic action of the intestines, and retards the onward passage of their contents, and thus permits of more absorption, if the lacteals and veins are in a condition to profit by it. When there is degeneration of the villi, or disease of the mesenteric glands, this will be of no avail as far as the particular diseased tissues are concerned; but the degeneration may not be general, and the retardation of the mass within reach of efficient absorbents may be so far beneficial. Again, digestion in the stomach is often stopped by too rapid contraction of that organ, and hence the intestines receive irritating food. In these cases a grain of opium given with each meal instead of in prescriptions may assist in keeping it within the stomach, and favour its chymification. Opium may be hurtful in cases in which by observation the liver secretion is seen to be diminished under its use, or in those in which it checks the action of the bowels, retains the evacuations long enough to permit their partial decomposition, and thus increases colic and flatulence; but as a general rule it will be found that two or three grains of opium, or even more, given daily, combined with ipecacuanha, or even without it, will not be hurtful. Most of the preparations of opium or morphia may be used. *Astringents.*—These are often very inefficient and sometimes add to the discomfort of the patient by causing flatulence. Nevertheless they should be tried. If there be acidity, the preparations of chalk should be prescribed. The less in bulk the better. Bismuth, soda and morphia, form a very useful combination. Nitrate of silver, the sulphates of iron, copper and zinc, with or without opium, sometimes answer; as do tannin, gallic acid, simaruba, kino, alum, the mineral acids, &c., which may be tried successively. One grain of tannin given every one or two hours has sometimes been beneficial, when the usual doses three or four times a day have failed.

In some cases when the Diarrhœa is checked, anasarca and ascites will be found to increase. It is not often, however, that we find that the flux will be stopped in such stages; but if it should happen, it would be well to encourage the action of the skin by sudorifics, and that of the kidneys by diuretics, and if these fail, astringent medicines should be reduced or temporarily withheld.

Chalk or other mixtures should never be made up with sugar and mucilage in the hot and rainy seasons of India, or they should be made up for the day only. In less than twenty-four hours the mixture runs into complete fermentation, and is hurtful in Diarrhœa. The chalk mixture of the London Pharmacopœia is always useless in India. It is better to order mixtures prescribed for the occasion, containing ten grains of chalk to each dose, to be sweetened if desired at the time of taking.

Diet.—This demands the greatest attention in all states of Diarrhœa. In the chronic states what is called low diet is not suitable. The more nutritious the food and the more that can be taken without aggravating the flux, the better; we are limited, however, to diet which shall be digestible by the feeble stomach, and which shall not irritate the canal through which it passes, and this compels us to attend both to quality and quantity. In the mild cases of feculent Diarrhœa, simple abstinence, or farinaceous food for a day or two, will be necessary. This affection is sometimes kept up by too much food. In the protracted forms of Diarrhœa the dieting is not such a simple matter. It is in these cases of primary importance that it should be thoroughly digested before passing through the pylorus. In order to know whether the food which is taken is digested, the evacuations should be inspected frequently, after washing the matters if they are pultaceous, and by scrutinizing the sediment when they are liquid. These examinations show what food has been assimilated and what not. All such kinds as have not been properly reduced should be avoided. It is sometimes better not to give the food in a too liquid state. Some people who have Diarrhœa find this lessened or stopped when they leave off drinking much tea or water. Beef and pork and all tough parts of meat should be avoided. Mutton, veal, chicken, pigeon and game will frequently be borne. They should not be overdone in cooking, but this is of less consequence if they are gently stewed. Eggs in a raw or half-boiled state are good. Milk is often an excellent food, but not so if the casein is found unchanged in the stools. Milk combined with lime-water or with farinaceous food or broth should be freely given if found to agree, and then it is most valuable. Many kinds of food which do not digest if given in large quantity, often answer very well if given in small amounts. Starchy foods, such as arrowroot and sago, often produce flatulence, and do not suffice for prolonged nutrition unless combined with milk. Rice should always be old and given immediately after dressing. When mixed with milk it sometimes answers well, but at times disagrees. It is the food of many millions of people, and who often crave for it, but care should be taken that it is old and well cooked.

In Indian hospitals the wheat-eaters should be provided with the best kinds of fresh ground-flour free from bran. Soojee or the internal part of the wheat grain should be given if possible, and when well cooked and mixed with milk is nutritious.

It is not a good plan to restrict patients to farinaceous food; those who are accustomed to animal diet should be allowed it if they can digest it. I have known the inner part of a mutton chop cause less irritation than a basin of arrowroot. When it cannot be taken in the solid form, animal food may be borne in the shape of strong soups, chicken boiled with rice, as in the Indian "pish pash," or made into tender stews. In some cases of Diarrhœa in young children raw meat acts very favourably. In one instance known to me, though it cured the patient, tapeworm apparently resulted. Occasionally good pepsine given with the food seems to assist digestion, and should be tried, especially, in the lienteric cases.

Wine and stimulants may be given, as required, both in acute and chronic Diarrhœas. Good port wine and brandy-and-water are perhaps the best for chronic cases. Acid wines and beer seldom suit the patient.

In young children attention to diet is of the greatest moment; a change of wet nurse is often enough to cure troublesome Diarrhœa in an infant, and change from hand-feeding to breast-milk equally useful. It is sometimes necessary to effect the change slowly and carefully; an infant allowed to take its fill will often take more than it can digest, and its Diarrhœa be increased. When a good nurse cannot be had, or the child is too old, donkey's milk may be given, or a goat should be kept, and, in warm climates, milked just before using the milk. Goat's milk generally requires to be mixed with one-third water.

For fear of increasing the irritation of the bowels, fruit and vegetables are often entirely excluded from the diet of diarrhœal patients. Such a course, if they are not already scorbutic, is likely to render them so. I have seen such effects. Fresh vegetables thoroughly and properly cooked, and soft fruits in moderation, should be allowed. Mealy potatoes, thoroughly ground down or mashed, are not hurtful, but unbroken lumps often entirely escape digestion, and cause irritation. If there be signs of scurvy, lime-juice syrup, pulp of grapes, ripe orange juice, or other antiscorbutic diet, should be cautiously tried.

It may be well to remark that the best of medical treatment of Diarrhœa may be rendered useless by bad cooking of the food. This is particularly the case in hospitals, especially in India, where the surgeon should consider the cooking very worthy of his attention.

Residence and Clothing.—Diarrhœal patients should remove from low and damp situations, and from dwellings subjecting them to malarious or vitiated atmospheres, to places in dry and open situations. They should be particularly careful of their clothing, and guard against sudden alterations of temperature. Flannel next the skin and flannel waist belts should be worn.

Change of Climate.—Change of climate for chronic Diarrhœa has long been understood and appreciated in India. I doubt not that it would be very beneficial in Europe also. In India there should be no hesitation in recommending this course, especially in the cases of white flux when the nutrition and strength begin to fail. Delay until emaciation and anæmia come on is very dangerous, as the chances of recovery are much reduced. Early and thorough change is more urgently required in old than in new residents. Should a patient after a month or six weeks find remedies unavailing, and that he is getting thinner without other cause than Diarrhœa, he should take change of air. The European may move away to some distant station in India, and the native of Bengal should go to the Upper Provinces, all seeking the places most free from malaria. The Hill stations are to be avoided. Patients near the coast should get to sea, which may be easily done, owing to the many coasting and other steamers plying in the Indian seas. These changes within Indian limits may suffice for recovery if carried out in the early periods of the disease; but for old Indians and for those who have been long ill, a change to Europe is imperatively demanded. The practitioner in India cannot too well understand that if this disease is not stopped it is generally progressive, although perhaps from day to day there is little perceptible change, and that the sufferer should not wait until the pearly conjunctiva, the general anæmia, the emaciated form, and the darkened skin warn those about him how firmly the disease has established itself. Nothing is more painful than to see an anæmic, anasarcous, feeble, diarrhœal patient being "sent home," as it is called. The sufferings on board ship for these helpless people can be well imagined. When undertaking a voyage, care should be taken that suitable provision and nursing should be provided; but this, though possible for people in good circumstances, is often impossible for poorer persons. There can be little doubt that being sent to sea without comforts in an advanced stage of disease is an aggravation of their condition, and that they should be spared this by judicious early travel. It is to be hoped that the time will come when all ocean passenger steamers will specially provide for the comfort and care of sick passengers. Cabins with extra attendance and good sick cookery would be a great boon for the helpless invalid from Diarrhœa and dysentery, and for this a moderate extra charge would cheerfully be paid.

Mere change to England is not all that is required. When there, there must be a continuance of the same principles of treatment, the same attention to diet, to dwelling and residence, and even more attention to clothing and providing against cold or chills.

Convalescence.—All chronic Diarrhœas show tendencies to relapse, and therefore there must be for a long time avoidance of exciting causes in all patients, especially in articles of diet, and many months should pass away before the patient may return with impunity to his previous conditions of living. This is particularly to be attended to as regards return from Europe to India. A severe chronic Diarrhœa in old Indian residents demands two or three years' absence from India for safety on return, even although convalescence should have been restored soon after arrival in Europe.

EPIDEMIC CHOLERA.

By Edward Goodeve, M.B.

Definition.—An epidemic, and in some places an endemic disease of great mortality. Typical Epidemic Cholera is characterised, in its developed stages, by vomiting and purging of watery fluid; by rapidly causing a state of the body called collapse, in which there is extreme depression or diminution of nearly all the functions of life; by terminating in death,—often within twenty-four hours from the first symptoms of the disease,—or in healthy reaction, or in various dangerous sequelæ, mostly of a typhoid nature.

Synonyms.—Cholera Morbus—Epidemic Cholera—Asiatic Cholera—Algide Cholera—Cholera Asphyxia—Cholera Spasmodica—Blue Cholera—Malignant Cholera.

The disease called by the above names is a severe epidemic affection, well known in India, and which has at intervals ravaged different parts of Europe, Africa, and America. In Calcutta and Bombay it prevails so constantly that it may now be said to be endemic there. It appears frequently in our camps and stations in India, and its occurrence therein, in violence, is a signal for unwearied toil and labour to our regimental and other surgeons, and of anxiety to all. And it may well be so, so widespread is its diffusion, so destructive are its effects, and so resistant is it to treatment. It demands, indeed, the careful study of all who practise medicine, and most especially of those who pursue it in our Indian territories.

History.—In a practical sketch such as this, it would be vain to occupy space and time with inquiries as to whether Cholera was, or was not, known to ancient writers. There is little room for doubt that our forefathers were acquainted with it in Europe. In India, Cholera has been observed several times since the English have had possession there. There is evidence of its having been known in Madras in 1769, 1770, and 1774. It attacked the artillery commanded by Colonel Pearse, marching to join Sir Eyre Coote, in 1781, and the troops under Colonel Cockerell, in 1790. Its occurrence in three or four places in different parts of India, in the beginning of the present century, is well authenticated. The epidemics, though apparently sometimes severe, do not seem to have been very frequent. Independently, however, of old records, we have a prominent starting point for the history of Cholera in 1817, since which time it has been frequently prevalent. In 1818 the Western world was startled with the intelligence of the appearance in India of a disease which was ravaging Lower Bengal, and had also attacked the camp of the Marquis of Hastings, then engaged in the Mahratta war, and who was at that time halted on the banks of the Sind, in the Upper Provinces. A new disease, or at least one unknown in such a terrific form, was carrying destruction through all the ranks of the army, both European and native. The scourge appeared in Lord Hastings' camp on the 6th November, 1817, and in five days destroyed 5,000 men. In it, in all, 9,000 deaths occurred. But not in the camp of war only did it cause surprise and terror. After having shown itself during the previous months in Mymunsing, Patna, Kishnaghur, Chittagong, and some other places, it burst out in August 1817, in the agricultural province of Jessore, among the peasants and labourers of the rice swamps and palm groves. Many thousands were swept away by the pestilence in the course of a few weeks. There might have been such a disease in the mist of past ages, but the memory of living man possessed no vivid or substantial knowledge of it. It burst upon the suffering generation with the violence of an unheard-of plague; impressing all with dread and consternation.

From this starting-point in India, Cholera spread east and west, far beyond the bounds of Hindostan. Its appearance in other lands may be traced with tolerable accuracy. From Bengal it spread eastward and southward in the following chronological order. We find it in

1818, in Burmah, Arracan, and Malacca ;
1819, in Penang, Sumatra, Siam, Ceylon, and the Mauritius ;
1820, in Tonquin China and China ;
1822, '23, '24, in all China ;
1827, in Chinese Tartary.

Turning to the west, we find it, in

July 1821, at Muscat and the Persian Gulf ; in
1822, in Persia, and prevailing there during 1822, '23, '29, '30 ; and in
1823, at Astrachan,

without spreading further westward for some years, *i.e.* until 1829, when it reached Orenburgh through Tartary, revisited Astrachan in 1830, and from thence started on its course through Europe.

The westward course continued slowly. In May 1831, it was very severe at Moscow and Warsaw; in July of the same year at St. Petersburg and Cronstadt; in October, at Berlin and Vienna. In England the first cases showed themselves at Sunderland, in October 1831, and the epidemic prevailed in the British Empire for fourteen months. It crossed the Atlantic and reached Quebec in 1832. This fatal malady ravaged the whole of Europe, and left that quarter of the globe in 1837; the last place affected being Rome. Since 1817 epidemics of Cholera have been frequent all over India, so that the disease may be said to have been naturalized there; causing a large mortality among all classes.

Besides the first great epidemic above mentioned, the western parts of the world have suffered from two severe visitations of Cholera, viz. in 1848-49, and in 1853-54. These appear to have travelled from the East much in the manner as that of 1832.

This Cholera seems to have spread east, south, west, and north from its first birth-place in Bengal, which became but the centre of an epidemic area comprising nearly all the world. It travelled slowly at first, and not continuously, but in irregular waves, checked sometimes, but not destroyed, by winter cold. Neither climate, nor season, nor earth, nor ocean seem to have arrested its course, or to have altered its features. It was equally as destructive at St. Petersburg and Moscow as it was in India; as fierce and irresistible amongst the snows of Russia as in the sunburnt region of India; as destructive in the vapoury districts of Burmah as in the parched provinces of Hindostan.

ETIOLOGY.—The predisposing causes of Cholera are doubtless common to some other epidemics. The exciting cause is, probably, an aërial, or at all events an air-borne poison, and probably of the zymotic class. We do not know, however, whether it is of an organized, organic, or inorganic nature. There are many circumstances in favour of its being of organic composition, some of its being organized. It has hitherto eluded all chemical and microscopic research. We are much in the dark as to its origin, preservation, multiplication, or diffusion. The poison is generally supposed to be some addition to the ordinary atmosphere, but some writers have speculated upon there being merely some modification of its ozone or electricity, or upon some dynamic change. Others have sought for an origin in telluric influences. In considering the causation of Cholera, we must bear in mind that we have to account for a disease spreading quickly over large areas, often preceded by epidemic diarrhœa; frequently developing itself with little warning in the places attacked, and often disappearing suddenly and returning to them after brief intervals, remaining absent for many years; visiting with great regularity the same places on each return; sometimes limiting itself with singular abruptness in certain localities, passing over places in its route with strange capriciousness, and afterwards returning to them, spreading sometimes with and sometimes against the direction of the prevailing winds.

It will be well, apart from all theory, to consider the various circumstances which appear to have some connexion with the development of Cholera. These may be divided into those which act upon the individuals in masses or groups, and those which belong to the individual only. To the first belong meteorological conditions, climate, soil, purity of atmosphere, food, water, &c.; to the second class, sex, age, occupation, &c.

Atmospheric Conditions.—No solution of the question of the cause of Cholera can be found in ordinary atmospheric changes. The opposite states of heat and cold, humidity and dryness, high and low barometric states, &c. have prevailed or been excluded without banishing the disease. A certain amount of heat seems to favour the spread and severity of the epidemics. In Bengal the hot seasons, including the hot and dry and rainy seasons, have witnessed the worst epidemics. In Bombay, Dr. Ewart's tables (Vital Statistics of the Armies in India) show that for eight years the most fatal cholera months for European troops were from April to September; the admission to strength, and the mortality to treated, being much higher: the percentage of fatal cases to treated giving 50·710 per cent. from April to September, and 19·510 per cent. from October to March: and in 3,676 admissions, during seventeen years, April to September gave 2,918 cases, and October to March, 758 cases. Dr. Morehead shows that the greatest number of admissions into the Bombay European General Hospital took place in the months from April to September inclusive. He mentions, however, that Dr. Leith gives rather different results for natives. In his tables of mortality in Bombay for the three years 1848–52, he reports 7,112 deaths from October to March, and 5,110 from April to September. Dr. Hugh Macpherson has shown that, in Calcutta, Cholera is always most rife in the hot months, both for Europeans and natives; the severity of the disease generally rising from January, through March and April, and descending from that month to August, when it is at its lowest. In the recently published Report of the Commission appointed to inquire into the Cholera Epidemic of the North-West Provinces of India, in 1861, it is shown that the curves of the disease for the North-West Provinces do not quite agree with those of Bengal, as will be seen presently. In Europe, temperature appears to have had an influence. In England, in 1848-49, the highest point of the curve line was in September. In 1853-54, Mr. Glaisher and others record that the greatest violence of the disease was during the months having the highest temperature. Although the greatest mortality in India is in the hot weather, there is a considerable amount of it in the cold seasons; much less, however, in the cold seasons of the North-West Provinces than in those of Bengal. In Europe also it prevailed somewhat severely in the winter of 1848-49, and very severely in Russia during the winter of 1830-31. The circumstance of the Russian houses being kept very warm inside hardly accounts for this. If Cholera were extinguished by cold, it is difficult to see how this action could have been neutralized in entire districts by the warming of the insides of the houses. It appears, from the above, that Cholera exists under very wide ranges of temperature, but that nevertheless it is probable that a certain amount of heat does contribute to its intensity.

More persons are attacked in the early morning than during the rest of the day. The temperature of the individual may be lowered during those hours, and this may have the effect of predisposing him to suffer. Possibly the lower temperature of the air may cause a greater concentration of the poisonous atmosphere near the ground at that time. The tendency of attacks to commence in the early morning has been noticed in Scotland by Dr. Adams, and in India by Mr. Twining, Dr. Morehead, Sir R. Martin, and others.

Rain and Moisture.—These do not prevent the spread of Cholera. It prevails most in Calcutta in the dry season. It does not do so in the North-West Provinces of India, in Bombay, or in Madras. The North-West Provinces have at times suffered severely in the hot and dry months, but the majority of the epidemics have been in the wet months, as shown by the Report of the Commission of 1861. Singularly enough, though the dry months are now the most fatal in Bengal, the great and desolating outburst, which startled the whole world, ravaged Jessore in August 1817, in the midst of one of the wettest seasons known. The combination of heat and moisture, when the air is moving slowly, or not at all, seems very favourable to the spread of Cholera. Thus a prevailing hot, moist, and stagnant atmosphere during these epidemics has been recorded by many writers. This is mentioned by Mr. Thom in his report of the memorable outbreak of Cholera at Kurrachee in 1846. He states that the dew point was very high, 83°, with thermometer at 90° in the shade; and there was induced a sense of languor and oppression, a stifled feeling about the respiration, and inability to undergo the least fatigue. This sense of languor and oppression is often felt during the lulls in the rains in India, independently of Cholera times, and therefore must not be considered causative of the disease. A warm, moist, stagnant atmosphere in Bengal, at any time of the year, is often followed by sporadic cases of Cholera, or by an increase of cases where the disease is endemic. Cholera has been known to cease after heavy falls of rain, and, on the other hand, to set in immediately afterwards, as in the Meerut gaol in 1861. In considering the influence of rain, we must recollect the temperature prevailing, and the time that has elapsed after the said fall. A very few hours after a fall of even heavy rain, if there be no wind, is sufficient to produce just the stagnant, hot, and moist atmosphere which is so oppressive to the feelings, and favours so much the spread of Cholera. One must not be misled by the name of dry months. The hot months in Calcutta are called the dry months, and are indeed the driest of the year, yet a great deal of moisture exists dissolved in the warm atmosphere; absolutely a greater amount of moisture in the air there than in England. In 1853-54 the atmosphere in England was drier than usual, for every month except May and December, the rainfall being 18·62 inches, or 5·93 less than the average.

Winds.—The only way in which these seem to influence the spread of Cholera is when they blow over places charged with miasma. It has appeared to prevail in certain situations when the air came across foul places, as privies, cesspools, &c., and shifts of wind have been known to have been followed by subsidence of the disease. It declined after a hurricane which took place in Madras in 1818. In Europe there was no fixed relation between the quarter from which the wind came and the intensity of the disease. Though Cholera appears to travel with the wind in many instances, it does not always do so. Orton reports that it travelled across a great variety of country, from the Nerbudda to Bombay, directly opposed by a strong wind blowing night and day for half the year, at the same rate of progress that it passed from Madras to Cape Comorin, with the breeze in its favour. Absence of horizontal movement of the air, or a stagnant atmosphere in combination with heat and moisture, has already been alluded to.

Electricity—Ozone—Barometric Pressure.—These have not been found to exercise any decided influence, or to have existed in different conditions during different epidemics. Most extended observations on some of these points, by Mr. Glaisher, are to be found in the appendix to the Cholera Report for 1853–54, and in the report of the Indian Sanitary Commission of 1862.

Much has been written on the influence of meteorological states upon the spread of Cholera, and there is much confusion and apparent contradiction in the results and on the inferences drawn. Some connexion between atmospheric states and the epidemics does exist, but it is not that of cause and effect. It is very probable that heat and moisture, which are so favourable to most organic actions and to chemical changes, may promote the multiplication of the Cholera poison, and that a calm stagnant atmosphere may allow of its concentration. We do not look upon heat and moisture as the cause of a tree or plant; but we find them very influential in their developments. Mr. Glaisher's observations in concluding his report on the Meteorology of London, in relation to the Cholera epidemic of 1853–54, allude to those of 1832 and 1848–49, and show that the atmospheric conditions during the prevalence of Cholera are well worthy of attentive study. He says: "The three epidemics were attended with a particular state of atmosphere, characterised by a prevalent mist, thin in high places, dense in low—during the height of the epidemic; in all cases, the reading of the barometer was remarkably high and the atmosphere thick. In 1849 and 1854 the temperature was above its average, and a total absence of rain, and a stillness of air amounting almost to calm, accompanied the progress on each occasion. In places near the river, the night temperatures were high with small diurnal range, a dense torpid mist, and air charged with many impurities arising from the exhalations of the river and adjoining marshes, a deficiency of electricity, and, as shown in 1854, a total absence of ozone, most probably destroyed by the decomposition of the organic matter with which the air in these situations is strongly charged."

"In 1849 and 1854, the first decline of the disease was marked by a decrease in the readings of the barometer and in the temperature of air and water; the air, which previously for a long time had continued calm, was succeeded by a strong south-west wind which soon dissipated the former stagnant and poisonous atmosphere. In both periods at the end of September the temperature of the Thames fell below 60°; but in 1854 the barometer again increased, the air became again stagnant, and the decline of the disease was considerably checked. It continued, however, gradually to subside, although the months of November and December were nearly as misty as that of September."

Climate.—We are not in a position to estimate the precise value of climate upon the intensity of the disease. The statistics of the native populations of India and of nations of the Eastern part of the globe are not sufficiently well known to enable us to compare the virulence of their epidemics with those of Europe. For India, we have only the military statistics on which we can rely, but they are of too exceptional a character to serve the purpose of the inquiry. Whatever may have been the virulence of separate epidemics, there is a much greater frequency of the epidemic in India. Whereas Cholera has visited Europe only twice severely since 1832, the epidemics are of constant occurrence in some parts or other of India. It is doubtful whether there is any relation between the marsh malaria of a country and Cholera. The question of the influence of climate will be again referred to in the section on mortality and susceptibility.

Nature of the Soil.—Cholera has prevailed so severely nearly all over the world, that it is not likely that the soil exerts much influence. Some writers have thought that it has been less severe on the Laterite foundations, in Madras and other places. Dr. Maclelland states that stiff clayey soils have had more mortality than loose sandy and easily drainable ground. Dr. Lorimer found that nearly half the epidemics examined took place on the black cotton soil. The volcanic formations of Auvergne escaped in 1832, although surrounded with Cholera fields. Dr. Farr states that in England it was less fatal on the primary geological formations than on others.

Elevation above the Sea Level.—The most favoured seats of Cholera all over the world are places not high above the sea, along the banks of rivers, and the estuaries of great streams. In London, the lower elevations were most fatal; and as the height of the locality increased, mortality decreased. Doubtless this is not from any difference of barometric pressure, but because these situations generally combine so many unfavourable sanitary conditions, as the most moist subsoil, the worst drainage, the least ventilation and air movement, the most impure air, and the most dense populations. In London and its neighbourhood the mortality was at the rate of 156 per 10,000 in the lowest districts, viz. Newington, Rotherhithe, St. George's Southwark, and Bermondsey, about the level of the Thames; and 15 per 10,000 in the highest, viz. Hampstead, Islington, Marylebone, and St. Pancras. There were some exceptions in places in which all sanitary conditions were perhaps worse than some of the places of a little lower level. Cholera is less prevalent in mountain elevations than on low levels. This is the case in the Indian hill stations. They have not, however, been exempt from severe visitations. Dr. Chevers mentions, in his "Review of the Means of preserving the Health of European Soldiers in India," that it prevailed in 1845 at Kussowlie, 6,000 feet above the sea, and at different periods at Murree, Dhurmsala, Dajeeling and Jackatalla, 6,000 to 7,000 feet high, and in lower elevations at Soobathoo, Hazeerabagh, and Mabaleshwur, 2,000 to 3,000 feet above the sea.

Impure Air.—There is abundant evidence that a foul atmosphere promotes the severity of Cholera. For copious illustration of this and the numerous sources from which the atmosphere may be vitiated, the reader is referred to the various reports of the Board of Health on the occasions of the English epidemics. Among others, the evil influence of privy emanations has been frequently noticed; and the same evil influence is obvious in the Report of the Indian Cholera Commission for 1861, in the instance of the epidemic in the cantonment of Meean Meer. On the other hand, it is found that in many epidemics some very foul and filthy places escape altogether. Thus, the filthiest parts of Lahore and Gwalior escaped, while the cantonments of Meean Meer and Morar, close to these cities, were ravaged by Cholera. The immunity of the filthy Ghetto at Rome is another instance. In some instances places with apparently the purest atmosphere did not escape, as for instance, in 1849, the healthy parts of Middlesex, Hertford, Buckinghamshire and Kent. Nevertheless, in spite of exceptions, the places in which the air is most vitiated from privies, cesspools, drains, decaying animal and vegetable refuse, or overcrowding and concentration of human emanations, are those in which Cholera has generally been most fatal and most widely spread.

Impure Water.—This, doubtless, plays an important part in Cholera epidemics, either as a predisposing, or, as some think, as an exciting cause. Dr. Snow thought that the poison was produced in the alimentary canal, and existed in the Cholera evacuations, and that these, through leakage of drains, cesspools, &c. contaminated water, which when drunk communicated the disease. Whether this be the manner in which Cholera is caused by impure water or not, it is certain that this promotes the spread of the disease. Strong evidence of this has been collected by Dr. Snow and Mr. Simon. The latter, in his report on the last two epidemics of London as affected by impure water, gives us a statement of the mortality among the consumers of water, supplied by two companies drawing their water from distinct sources, but distributing it in the same district, at the same time, and among the same class of people; the pipes of the two companies being laid pretty evenly in the same areas, in many places running side by side in the same streets, and the houses supplied pretty equally distributed. The Water Companies were the Lambeth Water Company and the Southwark and Vauxhall Company. The first drew their supply at Ditton, above the influence of the London sewage and tidal flux, ths last from the river near Vauxhall and Chelsea. The Lambeth supply was tolerably pure, the Vauxhall Company's very impure. The deathe in the houses supplied by the Lambeth Company were at the rate of 37 to every 10,000 living; in those supplied by the Southwark and Vauxhall, at the rate of 130 to every 10,000 living. The population drinking the foul water appears to have suffered 3½ times as much as that drinking the purer water. This seems to be an experiment as free as possible from error. The population submitted to the test being 400,000—500,000; the only ascertainable differences of circumstances in the two classes being in the kind of water supplied. The well-known instance of the sickness following the use of the water of a pump in Broad-street, Golden Square, affords strong evidence of the evil influence of water contaminated with cesspool drainage. Dr. Routh attributes the severity of Cholera in the winter in Russia to the drinking of water from melted snow collected from the immediate neighbourhood of dwelling-houses and on which the Cholera discharges had been thrown. Impure water alone will not necessarily produce Cholera. There are instances of freedom from the disease with bad water supply in the same way as there are with vitiated atmospheres already mentioned.

Bad Food.—Mr. Grainger, in his Report on the Epidemic of 1848–49, says, "Several marked examples were brought under my notice where violent attacks of Cholera were distinctly traceable to the use of putrid fish, bad pickled pork, decayed cheese, &c." Dr. Carpenter quotes Dr. Brittan as authority for the fact of an outbreak of Cholera and Choleraic diarrhœa among a number of school children, who had eaten plentifully of spoiled oysters, and by which eleven of the sufferers lost their lives. In the earlier days of the appearance of the disease in India it was thought that many attacks were caused by the eating of diseased rice. I remember the case of a gentleman who was attacked a few hours after eating hermetically sealed fish. In this class of causes may be included the noxious effects of purgative medicines given during Cholera. Numerous Indian writers recognise the mischief produced by these. I believe that this is not confined to the saline and hydrogogue purgatives only. I have seen milder purgatives followed by Cholera. Sir R. Martin and Twining caution us against administering during Cholera times any purgative medicines likely to operate in the early morning; *i.e.* about the time that the first symptoms of Cholera generally commence.

Regiments marching.—In Madras these seem to have been very liable to Cholera, as shown by Rogers, Lorimer, and Balfour. From Dr. Rogers' Report on the Asiatic Cholera in the regiments of the Madras army from 1828–44, this is shown both for European and native corps. Large bodies or regiments or parties of European recruits *en route* to join their corps have suffered greatly. On the other hand, treasure parties, consisting of smaller numbers of men, from ten or twenty to a maximum of 100 or 200 men, have suffered little. Thus in the European corps the ratio of marches attacked to marches unattacked has been 23·92 per cent.; in the small parties 2·72 per cent.; the officers being comparatively exempt. Dr.

Lorimer's reports go to prove that long marches were much more fatal than short ones, and that there is a ratio between the distances marched and the attacks. In Madras, in the native troops, the attacks were 46 per cent. between 600 and 800 miles, and 75 per cent. between 800 and 1,250 miles marched. Dr. Rogers attributes the difference of susceptibility to fatigue, exposure, crowding, and other predisposing causes which act more severely for reasons shown on large than on small bodies of moving troops. The adherence of Cholera to troops moving seems at variance with the beneficial effects so often experienced on moving troops out of infected cantonments in Cholera epidemics. The difference is probably due to there being less fatigue, less crowding, better conservancy, and more hopefulness and cheerfulness on the part of men leaving infected cantonments for a short time, than in the case of ordinary marches. It has been known that a regiment suffering severely from Cholera in camp on the march has lost it on getting into barracks. A remarkable instance of this occurred in the case of H.M. 63d Regiment, which suffered extremely during the greater part of its march from Poonah to Bellary, but entirely lost the disease in two or three days after getting into barracks in the unhealthy station of Bellary. In this case probably extreme heat in tents, overcrowding, and fatigue in the month of April acted as powerful predisposing causes. H.M. 86th Regiment, which suffered so severely at Kurrachee in 1846, had come off a long and fatiguing march from Upper Scinde.

Influences belonging to the Individual.—Sex.—This has but little influence. In London in 1849 the percentage of deaths to living was ·67 in the male and ·65 in the female; in lunatic asylums 6·4 for males, and 6·6 for females; the attacks 8·9 for males and 11·6 for females. In India we can depend on the returns of the European corps only, and the numbers are probably too small to be relied on to show the comparative liability of the sexes. Ewart's tables give privates 1·74 per cent., and women 1·58 per cent. admissions to strength, and among privates ·70 per cent., and women ·50 per cent. of deaths to strength. *Age.*—Mr. Grainger states that the liability to fatal attacks increases after the age of fifty in both sexes; the ages from five to forty-five having the lowest comparative mortality. In India in 1861 the influence of age was hardly perceptible. According to Dr. Gull, in England it was most fatal to those under one year and over fifty-five, corresponding, indeed, with the general tendency to mortality in England.

Residence in India.—It has been thought that Europeans of short residence in India were more liable to suffer than those of long residence, but the opinion does not seem to have been formed upon any well-ascertained facts. The Committee on the epidemic of 1861 investigated the matter for that outbreak, but they consider their data too imperfect for forming opinions. They say, "We can base no conclusions upon the figures shown in this table. The soldiers whose period of service was shortest apparently suffered the most. The proportion of deaths to strength falls from 7·7 per cent. among men of less than two years' Indian service to 3·5 per cent. among men who have served more than ten years. . . . The proper investigation of this subject must be left to future observers." It will probably be found that the influence exercised by length of residence in India upon the liability to Cholera is as little important as that of the other predisposing causes that have been already noticed.

Previous Health.—Both the strong and the weak are susceptible of Cholera. Mr. Thom states that some of the most robust men of the 86th were struck down at Kurrachee. There are often seen in the Calcutta hospitals, cholera-stricken, the stout, rosy, muscular Affghan, as fine specimens of men, in bone and muscle, as can possibly be conceived. Doubtlessly previous debilitating disease influences the mortality. It is not so certain that it greatly increases the susceptibility, though Dr. Morehead mentions that cachectic and debilitating diseases appeared to have had a strong predisposing effect on the attacked in the hospital at Bombay. Scurvy and diarrhœa have probably some predisposing influence. In Dr. Gull's report it appears that little predisposition was caused by previous disease, and Dr. Gairdner mentions that the post-mortem examinations in Edinburgh showed very little disease in the bodies of those who died there of Cholera. In India, in 1861, the sick in hospital furnished 13·5 per cent. of cases to strength, while the men in barracks afforded but 4·7 per cent.; but there were probably other causes than mere predisposition at work in this instance.

Habits.—I believe that these have less influence upon the susceptibility to Cholera than has been supposed. Persons in whom habits of intemperance have established organic disease of liver or kidneys may be less liable to recover when attacked, but the real degree of susceptibility engendered by intemperance has not been completely ascertained. In India in 1861 the intemperate were more subject to attacks than the abstemious, but their chance of recovery in the attack was better. However, the numbers tested were small.

Occupation.—This seems to produce no special liability; but of course those which expose the individual to unhealthy influences and residence may increase his risks. Thus the privates and non-commissioned officers of regiments suffer more than the officers. Fatigue, want, grief, fright, have doubtless some degree of predisposing influence, though it would be difficult to estimate the amount in figures. Of these, fatigue is probably the most injurious.

Probably most of the causes mentioned above may be considered as predisposing causes. Impure air and water may convey the exciting cause, but this will be presently referred to. None of these in themselves appear to be sufficient to produce Cholera, though they may all render it more fatal. The existence of all the conditions mentioned, both before and after Cholera epidemics, without producing the disease, shows that some special agent or cause must be present to give rise to it. Before entering upon the question of this specific cause it will be well to mention some points in the natural history of Cholera not yet spoken of.

Health of Communities before and during Cholera Epidemics.—In Europe a prevalence of several zymotic diseases has been noticed in these periods; particularly typhus fever, influenza, and diarrhœa. The increase of fluxes was so great previous to the outbreak of 1848 that Cholera in England was looked for long before its arrival. There was also an increase of typhus fever. Dangerous and fatal influenza preceded the epidemics of 1832 and 1848. Diarrhœa is constantly present during Cholera. In London, for the quarter ending September 30, 1849, notwithstanding the large mortality from Cholera, the deaths from typhus fever were nearly equal to those of the four preceding corresponding periods; themselves of remarkably high mortality. In India, diarrhœa constantly prevails during epidemics, but the precedence and concurrence of other zymotic diseases is not so well made out. Cholera, however, does not seem to banish them.

From the above we seem justified in inferring that circumstances similar to those which favour Cholera promote also the spread of the other zymotic diseases.

Limitation of Areas of Cholera.—This is sometimes singularly abrupt. It has been known to attack persons on one side of a street, of a camp, or a town, only. In 1848–49 it attacked one side of a small village in Argyleshire, and confined itself exactly to one side of the town, which consisted only of one main street, divided into two equal parts by a toll bar, beyond which to the west not a single case occurred, though the inhabitants were in constant intercommunication. Dumdum, seven miles from Calcutta, often suffers severely, while Calcutta does not, and *vice versâ*. One part of a ship may suffer and the other not. A wing of a building may be ravaged while the rest escapes. The limitation to districts is sometimes remarkable, and was shown in the case of the 9th Lancers on its passage up the Ganges in 1842. The regiment travelled in boats, by wings, and at separate times. When the left wing reached Monghir, although there had been no communication with the shore, Cholera broke out in it, and continued with the detachment for about twelve days, until it had got beyond the infected districts. When the right wing reached Monghir, it, also, was attacked, and lost the disease exactly at the same place that the other did. The limited area of infection has been tested by the good effects of moving troops into camp when suffering from the disease. Numerous instances of this are on record; among the latest some are afforded in the Cholera Commission Report for the outbreak of 1861. A move of a few miles is generally sufficient.

Mode of Invasion of Localities.—We find that both in India and Europe it is often preceded by diarrhœa, sometimes for months, as in England previous to the outbreak of 1849. Then a few cases of Cholera appear, and in a short time the epidemic sets in severely. At other times the extreme violence shows itself rapidly and suddenly after the prevalence of a few cases for a few days, as at Kurrachee. Of this outbreak Mr. Thom says: "It suddenly burst forth in a few hours in every European regiment, whether in camp or in barracks, in every tent and in every house, and it was at its acme in forty-eight hours afterwards, when instead of spreading further it gradually and steadily declined. Now, it appears that for some days or even weeks a few cases had appeared in the native town of Kurrachee, but there also, at the same period, the malady became *suddenly* general over the whole place." In Paris, in 1832, Dr. Baly relates, that in eighteen days from its commencement it had reached its climax, and had already extended to all the quarters of Paris, and had been fatal to 7,000 people. In Lord Hastings' camp in 1817, 5,000 people died within the first five days of its appearance.

Departure of Cholera Epidemics.—These often leave a place rapidly, sometimes after sudden atmospheric changes. At Dumdum, in 1859, Dr. Hugh Macpherson reports that a violent outbreak occurred and carried off one-sixth of a detachment of recruits and their families in ten days, confining itself entirely to the barracks; it then disappeared as suddenly as it came. In the 2d Madras Europeans, as before mentioned, the disease continued for a month in hot and sultry weather, and disappeared, also, as suddenly as it came. The occasional sudden departure after high winds and storms has been already alluded to. Generally the decline of Cholera is more gradual, the intensity of the mortality diminishes, the recoveries to attacks are more numerous, and finally the disease disappears, In England in 1848–49 the epidemic was about three months from the commencement of its decline to its final disappearance from the country; having prevailed about sixteen months. Cholera is apt to leave a place for a time, and then return to it, and be as severe on its second as on its first visitation. In 1848-49, Cholera prevailed slightly at Sunderland from October to January, was absent for a month, returned with severity, subsided for two months, and returned a third time with still greater violence, The epidemics do not break out simultaneously all over a country, but there is, nevertheless, often the beginning of the disease in several distant places at the same time; thus, the epidemic of 1848-49 first showed itself on the same day in Edinburgh, Sunderland, and Hounslow. It broke out at Malta, Palermo, and Gozo at the same time. It appears that Cholera epidemics have a tendency to reach their climax over large areas at about the same time. Dr. Baly states that the period of the greatest intensity of the epidemic of 1848-49 was, in the majority of cases, in August and September; thus, of 226 places affected, 61 suffered most in August and 118 in September, or 179 out of 226, in those two months. Also that in large towns or cities there was the same general approach to simultaneousness, as appeared in a comparison of different counties and of different towns one with another. Occasionally in epidemics a smaller climax is noticed, and there is also a simultaneousness in the period of this; that for 1848-49 was in January 1849, when a decline in the intensity of the disease took place pretty generally.

Cholera does not spread uniformly over a large area or country; it seems rather to spread from certain centres first attacked, often passing over places in its apparent course, and perhaps subsequently invading them. Some places indeed, although in the track, escape altogether. It has a tendency, even after long intervals, to return to certain haunts in the same towns; thus, certain places which suffered greatly in 1832 were equally attacked in 1848-49, and some remarkable coincidences occurred in illustration. It has happened that the very first house invaded in 1832 gave the first cases in 1848.

Protection by previous Attacks.—These do not appear to confer any immunity; there are numerous cases on record of persons who have had Cholera more than once. Some writers have speculated upon the susceptibility of individuals being diminished by long exposure to Cholera atmospheres without being attacked. The opinion does not seem to rest on any good foundation.

Channels of Introduction into the System.—Being ignorant of the nature of the poison, we cannot well say how it enters the blood; it probably enters through the respiratory or intestinal surfaces.

Incubation.—It seems that this may be very short for many cases; perhaps two or three days, or even less. Of course if we include the Choleraic diarrhœas and those cases which show other preliminary disturbances, mentioned hereafter, we must allow a much longer time. In the case reported by Dr. Barry, and referred to in the paragraphs on Contagion, the period of incubation does not seem to have exceeded forty hours. When Cholera breaks out in ships at sea several days after leaving port, the attacks are probably due to freshly acting causes.

Mortality to Populations.—This varies for different epidemics and in different districts. In 1848-49, in Dr. Baly's report, we find that the deaths were 30 to every 10,000 living in England and Wales. This mortality was not evenly distributed: the inland districts giving 17 per 10,000, and the coast districts 50 per 10,000, and 404 districts 7 per 10,000, and 85 districts in which there were none. The denser the population to the square mile, the greater the comparative mortality; thus a population of 915 to the square mile gave 65 deaths per 10,000, and 235 inhabitants, 7 per 10,000. In India among the European troops, according to Ewart, the deaths to strength of the men for 7 years was annually 0·70 per cent., or 70 per 10,000; of the officers, 0·12, or 12 per 10,000. It is not possible to ascertain what is the mortality of the native population of India from Cholera or any other disease. The susceptibility of populations is very difficult to ascertain, because a large number of attacks not fatal are never reported, and therefore, except in the case of troops, no reports are to be depended on. In India, Dr. Ewart places the liability of European troops to attack at 174 annually per 10,000 for the men, and 73 annually per 10,000 for officers. In India the percentage of attacks is much higher for European than for native troops. The Cholera Commission has shown that the native prisoners in gaols have a liability nearly the same as the European troops.

Diffusion of Cholera.—This is of the greatest interest. Setting aside for the present the question of contagion, it will be well to consider the facts of the diffusion of Cholera by human intercourse. From the mass of evidence on this point, it is impossible to doubt that in many instances it has been spread, in some manner, by such means. Dr. Barry, in the Indian Annals, for 1854, relates that Cholera made its appearance in the military hospital, at Gowalparah, on the 27th April, and that several cases afterwards occurred. The first case was that of a sepoy who had just arrived with a detachment from Gowhatty. Now, there was no Cholera at Gowhatty when he left, and none at Gowalparah when he arrived; but the whole detachment had, forty hours before, passed through a place called Palasbarree, where it was raging. It is supposed that the sick man brought the disease with him from thence. It spread, but not fast, and the first cases which occurred at Gowalparah were from those who passed through Palasbarree, their comrades who waited upon them in hospital, and the sick soldiers in the hospital into which they were received. The dispersion of the children of the Tooting school on account of the fearful outbreak of Cholera among them, in 1848, was attended with attacks and deaths among the children removed, and seizures among the inmates of some of the establishments into which they were received, although there was at the time no Cholera in the institutions or surrounding neighbourhood. In the Report of the Indian Cholera Committee on the epidemic of 1861, some remarkable facts are stated with reference to the introduction of Cholera into Gurwhal and Kumaon in 1852. The Report says, "The districts of Kumaon and British Gurwhal lie entirely within the Himalaya mountains, on the borders of Rohilcund. They cover an area of 12,000 square miles. The population is very scanty, scattered for the most part in small villages, which are often separated from one another by vast mountains and tracts of forest. These districts are cut off from the plains of Northern India by an uninhabited belt of forest, and by the swampy and almost deserted region called the Terai. These tracts, some twenty miles in breadth, effectually cut off the inhabitants of the mountains from those of the plains. The intercourse between them is at all times very little, and confined to a few particular lines leading to places of pilgrimage or trade. Cholera is generally as completely absent from these mountains as from any part of Europe, but it has occasionally spread among their inhabitants epidemically with great violence. Mr. J. Strachey, C.S. the able president of the Cholera Commission, formerly in civil charge of the Hill District of Gurwhal, mentions the following facts, which occurred under his own immediate observation. "In the early part of 1852, extensive works of irrigation were in progress at the foot of the mountains in Kumaon, in the Bhabur" (the strip of forest land which divides the mountains from the plains), "of which Mr. Colvin has spoken in his note. Several thousand workmen were collected there from all parts of the neighbouring hill. Cholera broke out among these people with great virulence, and they fled panic-stricken to their homes, which were generally at a distance of several days' journey in the interior of the hills. Up to this time Cholera had been unheard of in Gurwhal, or in any part of the neighbouring mountains. This is a fact which was carefully inquired into and thoroughly ascertained. Many of the workpeople who had fled from the Bhabur died upon the way to their homes; many others were attacked when they reached their villages. Then Cholera broke out among the other inhabitants of the villages, commencing in very many instances in the families of the men who had brought the disease from below. For a considerable time Cholera was entirely confined to places which had been in direct communication with persons suffering from the disease, but in the course of a few weeks it had become impossible any longer to trace such connexion, and Cholera became generally epidemic in the hills. Many of the first cases were carefully investigated; it appeared to be proved, beyond the possibility of a doubt, that in many instances Cholera had never been heard of in the villages until the arrival of the men from the Bhabur, who were the first attacked by it. . . . There were no other circumstances that could be discovered which appeared to throw even the possibility of doubt upon the fact that Cholera was brought by human intercourse into a district which, up to that time, had been perfectly free from every sign of the disease." There are other instances brought forward by Mr. Strachey, communicated by Mr. B. W. Colvin, C.S. and Dr. F. Pearson, Superintendent of Vaccination in Kumaon, and to these the reader is referred.

Although it is probable that Cholera is spread by human intercourse, it is indisputable that it originates in places without it being possible to trace any previous communication with infected persons. This has happened over and over again in towns and large establishments in which the outbreaks, after the greatest perseverance, could not be accounted for by any previous exposure to infection. Its appearance at St. Kilda, in the Western Islands of Scotland, is as strong an instance as can be given. This island was cut off from all communication with the mainland, and yet the disease appeared in it suddenly without a trace of importation. In 1848, Dr. Parkes could not trace any contagious origin for the first cases occurring in London.

Contagion of Cholera.—The fact of the diffusion of Cholera by human intercourse leads us to inquire how this operates, and to the question of the Contagion of Cholera. Diffusion by contagion does not negative the possibility of an origin independent of infection. The majority of medical men in India, accustomed to see Cholera year after year, to be in constant intercourse with the Cholera sick, and to see the general immunity of hospital attendants and of themselves, doubt the contagiousness. Some physicians, however, think differently. It does not seem proved that contagion can exist in the shape of a volatile poison emanating from the sick and rapidly infecting the healthy. It would not be so easy to disprove that there may not be some form of poison which may not be volatile, or which may require time to become so, or to develop its poisonous properties and capability of infection. A volatile poison, at all strong in its action, would be most dangerous to all about the sick, and yet in India the medical men, nurses, hospital coolies, sweepers, and others who are constantly engaged about the sick, do not appear to be more liable than the rest of the population. The disease seldom spreads from bed to bed in a ward; on the contrary, when people are attacked in hospital they lie generally in a distant corner, or in another ward. I have noticed this over and over again; and though I have been connected with the large hospital of the Medical College at Calcutta for many years, I do not recollect any spreading to the nearest or neighbouring patients. I should, as far as my own experience goes, say that Cholera does not spread from the sick to the whole by any rapidly acting emanation. Dr. Morehead's observations support the view of the non-spreading of Cholera in hospitals through contagion. They were carefully conducted through three epidemics in Bombay; and though he refrains from drawing positive conclusions, his facts are not in favour of contagion. His observations will be found in his valuable Clinical Researches on Diseases in India. Sir Ranald Martin states, that of the five native keepers and washers of clothes of the Calcutta European General Hospital, who during twenty-five years had kept and washed all the hospital clothing, not one had Cholera, nor had those who assisted them. The same may be said of the dressers and sweepers. In England, in spite of some suspicious instances, the washers of soiled linen and bedding do not seem to have suffered out of proportion to others. In Dr. Baly's report, he analyses the reputed cases of contagion from this cause, and shows that out of thirty-five reported cases seven only seem to support the contagion theory. It is remarkable that the washers of large collections of linen, soiled by cholera patients, in many large hospitals, did not suffer seriously in 1849. Dr. Waller Lewis's report to the Board of Health, in 1849, shows the error of the assertions of contagion as regards the case of a number of washerwomen reported to have caught the disease by washing soiled linen, and whose cases he personally investigated.

Against the foregoing may be quoted the cases reported by Dr. Barry before referred to; also the Report of the Committee of 1861, which records that, as far as that epidemic was concerned, the patients in hospital did suffer more than their proportion of attacks. Out of hospital the cases to strength were 4·7, the deaths to strength 2·9, and deaths to treated 62·7 per cent. In hospital, among patients, the cases to strength were 13·5, the deaths to strength 10·6, and the deaths to cases 78·5 per cent. This applies to the whole of the troops attacked with the epidemic in thirteen stations, omitting Meean Meer and Morar, in which the proportions were higher than the above. Including these stations, the cases to strength were 14·7, the deaths to strength 11·6, and the deaths to cases 79·2 per cent. So that it is remarked by the Commission that "the virulence of the disease among hospital patients was clearly more than twice as great as it was among the healthy strength of the regiments." The medical officers escaped entirely, and subordinate medical establishments, which in such epidemics may be said almost to live in the hospital, and the hospital servants, who must have been much more in immediate contact with the sick than the patients were, suffered but slightly; and of thirty men of the 31st Native Infantry sent daily to attend the sick from the 24th of August, when the epidemic was at its height, not one was attacked. It is not very easy to explain these discrepancies and the difference between the spreading in hospital in this and other recorded epidemics. As regards the comparison with the men out of hospital, there may have been something in the situation of the hospitals themselves to account for the disease spreading so much in them. It appears that in several instances the epidemic broke out first in them. It is possible that the number of patients exposed was too small to render the inferences drawn from their suffering free from fallacy. The returns of the particular regiments show that the greatest excess of difference was in those in which the patients in hospital were small, say 3 to 20. The ratio in those in which were the two largest numbers, viz. 113 and 117, was different; one being above and one below the numbers attacked in the lines.

Infection does not seem to have arisen from the dead body, if one may judge from the impunity with which an immense number of post-mortem examinations have been made in most places, and the contents of the intestines submitted to all kinds of examination. Drs. Mackintosh, Aitken, Gairdner, and others bear testimony to this.

Portability of the Cholera Poison.—A strong instance of this is related by Dr. K. Mackinnon in his work on Public Health, &c. "A regiment proceeding by water down the country had the disease badly. It met a corps coming up the country, with which it exchanged boats: the disease stuck to the boats, left the corps it first affected, and attacked the new regiment which had a clean bill." The breaking out of Cholera in ships many days at sea seems also to show that the poison may be carried. This does not necessarily imply that it was thrown off by a diseased individual. For aught we know, the poison in any district may adhere to surfaces of any kind, and be carried about or remain attached to walls, &c. until fitting circumstances call it into action. When attached to moveable surfaces, it may be transported to distant places, and be one of the means of diffusion through human intercourse. We have no knowledge of the period of the disease at which contagious emanations from the sick, if there are such miasms, are evolved. Those who believe that the poison is to be found in the evacuations would consider it to be given off with the rice-water stools. Dr. W. Budd informed me of a case which, he thinks, bears upon the question of the possibility of infection in the convalescent stages. A gentleman was attacked with Cholera in London, and in early convalescence came down to Bristol and was nursed by his mother, with whom he lived in a very airy, open, and elevated part of Bristol. In three or four days the lady was attacked and died. There was no Cholera in the neighbourhood at the time, though there was some in distant parts of Bristol.

Of late years Dr. William Budd, of Clifton, has advocated the contagiousness of Cholera through the medium of the intestinal discharges. His propositions are these:—1st. That the disease is essentially contagious or communicable; and 2d, That it is disseminated, as he believes, exclusively by the liquid discharges from the intestinal canal of Cholera patients. Dr. W. Budd believes that the poison is rapidly multiplied in the human body, and that the rice-water discharge contains the product of this multiplication and becomes a source of infection, so that from one Cholera patient virus enough may arise to propagate the disease to numerous persons. He considers that the poison may be disseminated in the following principal ways:—1st. By the soiled hands of attendants on the sick, a mode of communication which is probably very common within the limits of the family circle; 2d. By means of bed and body linen, and other articles tainted with the rice-water discharges; and 3d. Through the medium of the soil, which, as the discharges are liquid, necessarily receives the great bulk of them.

Dr. W. Budd thinks that from these places of deposition the poison may spread itself by rising into the air with the products of evaporation, by percolating into drinking water, or by atmospheric dispersion, in the form of impalpable dust, after it has passed into the dried state. *He considers that it is a disease which infects the ground.* He contends that the poison may be preserved for months or years in a dry state, in the same way that the germs of fungi or infusoria may be, and that they may in favourable conditions be brought into activity, and being received into the human organism, after a very short incubation produce the disease, and by multiplying with enormous rapidity provide sufficient material for extensive infection. On the other hand, he thinks that in unfavourable circumstances the germs may as rapidly decay and become extinct, and that thus an epidemic may end. In short, Dr. W. Budd imagines germs capable of preserving a permanent dormant vitality with susceptibility of immense power of reproduction in the living body, and liable to rapid decay when placed in unfavourable circumstances. These views are very simple; they are by no means far-fetched or beyond the analogy of other epidemic diseases, and have been received with great respect in England. They are supported by strong evidence, mainly of this nature:—Cholera has been known to rage violently among, and to be confined to, one sex in establishments containing both male and female inmates under the same roof, separated from each other merely by walls or partitions, and breathing the same air, eating the same food, and drinking the same water; the only difference between them being in the use of separate privies; the contamination of one of these privies by discharges thrown into it from the first case explaining the reason of the difference of suffering in the two sets of persons.

Although, according to Dr. W. Budd's view, Cholera is contagious, it is so in a very different form and manner than if it were so by means of a volatile or gaseous emanation proceeding from the sick. Doubtlessly it explains the diffusion of Cholera in a more satisfactory way than the ordinarily received notion of a contagious poison, and after the facts brought forward by him it would be wrong to neglect the practical application of their teaching. I think, however, that more evidence is required before it can be held to be proved that the specific exciting cause of Cholera is to be found in the discharges only. The theory will hardly explain all the phenomena of the spread of the disease in all cases. Take, for instance, the rapid appearance of the disease in the 2d Madras Europeans, mentioned by Rogers; its rapid diffusion at Kurrachee, in 1846, and in Paris, in 1832, as before stated; or such a case as this, mentioned in Jameson's report: in a cantonment perfectly free from disease, ten men of the same regiment were attacked in a single night, and every case proved fatal—no fresh case happening in the corps or any other. It is not to be supposed that these men were the only susceptible persons in the whole regiment. It may be difficult to account for the outbreak on any theory, but not less so on Dr. W. Budd's than on any other. It appears unable to account for the manner in which Cholera becomes milder in the decline of an epidemic. The deaths to cases are much diminished. We should expect that as the epidemic advances the virus would be more in quantity, more concentrated, more virulent; but such is not the case. The susceptibility in the population remains to a considerable extent, because numerous cases occur, but they are milder. We should expect that the virulence of the disease would go on increasing until the susceptible people were quite exhausted. Some other explanation than decay or partial decay of the Cholera germs seems needed. The regularity with which, over wide areas, Cholera epidemics advance, and the simultaneousness with which they attain their maxima and their decline, seem to imply some general law, and not a diffusion dependent upon the chances of fæcal contamination only. I think that those who have been in the habit of seeing much of Cholera will not be inclined to think that there is much potency at all events in the *fresh* Cholera discharges. It has been my lot, in numerous epidemics, to see many cases at a time, and for a long time in my own wards; to see the beds, the sheets, the hands of attendants, the floors, frequently—nay constantly—soiled with discharges; to see the utter impossibility of providing fresh beds and fresh blankets, &c. for every case; to see abundant opportunities for the diffusion of the poison from the discharges: and yet, as mentioned under the head of contagion, not to see Cholera spread in the wards. If fresh Cholera discharges were so virulent, every bed next to a cholera-bed would be a bed of the disease; every bed and metal bed-pan would be a source of Cholera to every succeeding patient.

My own observation is that no such evil results. The hospital sweepers, whose special duty it is to remove all the discharges, do not suffer out of proportion to others. Dr. Baly's report on this point has been already referred to. It may be that decomposing Cholera discharges may be more mischievous, as has been supposed by Pettenkofer. If, as supposed by Dr. Budd, the poison is multiplied so rapidly in epidemics as to suffice for the rapid diffusion which takes place in severe epidemics, it ought to be equally powerful when under observation in a sick ward. The mode in which Cholera spreads in wet weather in India seems adverse to its diffusion by the discharges. In dry weather, with a strong wind, it is easy to suppose that the Cholera dust may be spread to a considerable distance; but in the rains of the tropics and bordering countries we cannot trace this mode of diffusion. All matter infecting the ground must be swept away in the violent falls, to which English rain is but a trifle; it would, one would suppose, be diluted and swept away into rivulets and streams, and ultimately into great rivers. Sometimes it might overflow tanks, but in many parts of Upper India the drinking water is taken from deep wells; and where there are tanks, the Hindoos especially are careful to keep them pure, so that it is not so probable as may be supposed that they are often contaminated. And yet it is in the rainy seasons of the North-West Provinces that the severest Cholera epidemics have raged. Although the specific poison of Cholera may not exist in the rice-water stools, it must be admitted that Dr. W. Budd has brought forward strong evidence in support of his views, and the facts which he adduces can, in the present state of our knowledge, be explained only upon the supposition that the evacuations of Cholera patients have, in some state or other, an influence upon the spread of Cholera. There are yet many points that require elucidation to prove that the Cholera poison multiplies in the human body, though perfectly in accordance with the cases of many other epidemic diseases, so that the specific agent is contained *pur et simple* in the rice-water stools. It would seem that they are the congenial soil for the multiplication of the poison, rather than the direct source. It would be a good thing for mankind that Dr. W. Budd's views should prove correct. It would then be a comparatively easy thing to prevent the spread of the dire pestilence.

Cryptogamic Theories of Cholera.—Chemical analysis of the air has thrown no light on the cause of Cholera. It is possible, however, that prolonged microscopic examination of strained air, as in the method of Pasteur, might be more successful. Dr. Cowdell has advocated the cryptogamic origin of the disease. Dr. Britton made some careful examinations of air collected by an apparatus from Cholera rooms, and discovered therein some bodies which resembled others found by Dr. Swayne and himself in the rice-water discharges, and by Dr. W. Budd in the water of districts infected with Cholera. These were afterwards shown by Mr. Busk to be starch-granules, and a species of uredo or blight. Mr. Rainey, Dr. R. D. Thomson, and Dr. Hassall examined the air in the epidemic of 1853-54. The two former made an extensive series of observations at St. Thomas's Hospital. Although all these observers found organic forms in abundance, they did not discover any special cause for Cholera in those organisms. Nevertheless, there seems much that is attractive in the cryptogamic theory of the disease, and the whole subject derives additional interest from the late researches of Pasteur. Valuable information on this subject might be afforded by investigations made in India during the presence and absence of Cholera epidemics. The writer of this has often regretted that he never had leisure to undertake such investigations. Many of the phenomena observed during the march of Cholera epidemics might be explained much more satisfactorily upon the supposition of the exciting cause being masses of organisms moving in obedience to atmospheric impulses and currents, than by most other theories. They might multiply wherever they found a fitting nidus, which might be in privy atmospheres, or in air abounding in emanations from decaying and putrefying matter, or in crowded rooms, and, indeed, all vitiated atmospheres. They might appear to impart an infecting character to the choleraic discharges by multiplying enormously in them.

After allowing all possible latitude to the dissemination of Cholera by human intercourse, which may include the diffusion by gaseous emanations acting directly through the atmosphere, or through fomites, or the diffusion through the Cholera evacuations, it appears that we are still unable to explain all the facts of its origin in a place, and its spread over the land. Summarily we may say that we shall still want an explanation of those facts which show that Cholera will arise without the possibility of tracing any communication with the sick, or with fomites or choleraic discharges. It cannot be said that the supposition of a poison, cloud, or miasm, spreading through the land, will account satisfactorily for all the phenomena, either such as its strange limitation to certain areas, or its passing over a town on its apparent track, leaving it unscathed, or nearly so, to show itself violently in some place beyond the town so spared. The only way in which this capricious action could be accounted for, would be on the supposition of clouds of matter or of fungi or infusoria, limited in their spread by atmospheric currents, sometimes compressed into vertical strata and touching the earth in a thin line or belt only, or lifted upwards by some upward atmospheric movement, and thus being above the places passed over. But though it would be possible to conceive this mode of progression of a mass of organisms obeying the impulses of the atmosphere, yet no such organisms have been hitherto demonstrated, and, indeed, the progress of the disease against strong periodical winds would seem to oppose the view. May it not be a mistake to consider the specific cause at all as a simple body, either generated from without, and air-wafted to a particular spot, and then multiplying itself indefinitely, or as a locally-generated agent, and spreading over certain areas? Might it not be more in accordance with facts to suppose that neither a miasm from without, nor a miasm from within, exclusively contains the specific poison? Might it not be that two factors are needed, the one some air-borne material or some dynamic modification of atmospheric elements coming from without, the other some local element: neither being potent unless united? The peculiar atmosphere sweeps along hither and thither, and it is only when it meets with the other peculiar substance that the poison is generated. It may be that the Cholera evacuation is the most prolific of the peculiar local agents. Some general, not local law, seems to govern all Cholera epidemics. Contagion from Cholera discharges may operate, but there must be something beyond this. Contagious diseases are not epidemics at all times, even in the same places. In Lower Bengal every year the native small-pox inoculators proceed to inoculate patients. The disease in these cases rarely spreads. For years successively this may go on without evil results, and there is no uncontrolled diffusion; but every few years a wide-spreading epidemic of small-pox, which nothing seems to resist, sweeps over the land. There is nothing discoverably different in the epidemic and the common years—the same people, the same habits, the same places, the same filth, or the same absence of it. In all the seasons there is the poison, in all the seasons there is the susceptible population; and yet how different the results! It is not to be supposed that the mere amount of poison generated in times of confluent small-pox makes the difference; but even if so, how is it that in one year all the cases are confluent, in others discrete? Why has the inoculated poison such a limited influence in favourable years? If there be poison in the Cholera stools, it may bear the same relation to Cholera epidemics that the inoculable virus of small-pox in healthy years bears to the destructive epidemics of the exceptional ones; in certain circumstances a sufficient cause—not the only one.

Though it must be confessed that we do not know what is the exciting cause of Cholera, we may hope that we are grasping some of its laws. It is impossible to deny that it riots far and wide, independently of contagion; but we must admit that it may be spread in some way by human intercourse. This is perhaps more satisfactorily explained by the views of Dr. W. Budd, than by other modes of contagion. The influence of predisposing causes is certain, and it is undoubted that they may be rendered less active. It is to be hoped that the Sanitary Boards now permanently given to India by the recommendations of the Royal Commission on the Sanitary State of the Army in India, will do much to diminish the mortality of Cholera. While grateful to them for their labours and recommendations, we must in justice to the officers of the Royal and Indian Medical Services state, that many of them had earnestly striven in the cause of preventive medicine long before the Committee was formed, and that most of the facts published by it were well known and appreciated by the said services. Among those who have worked in the spirit of the Sanitary Committee, and who have done much to advance the cause of sanitary science in India, either by their writings, by their position about those high in power, or by their official standing or appointments, Kenneth, Mackinnon, Martin, Bedford, Chevers, Parkes, Balfour, Morehead, Rogers, Lorimer, Maclean, Alexander Grant, Forsyth, Hugh Macpherson, Ewart, Maclennan, Leith, Mouat, and P. Walker may be mentioned with special honour.

SYMPTOMS.—The typical cases of well-developed Cholera present well-defined symptoms. The descriptions in the following paragraphs apply to the ordinary forms of Epidemic Cholera. In the beginning of an attack, the most prominent symptoms are disturbances of the stomach and bowels. Then, in swift succession, though not necessarily in that order, the circulatory, respiratory, muscular, and nervous systems suffer. Then, in severe cases, ensues extreme depression of all the functions of life, which often terminates in death. The symptoms are naturally divided into periods or stages, which may be called those of *Invasion*, *Development*, *Collapse*, and *Reaction*.

Stage of Invasion.—We have not very often to deal practically with more than one or two sets of symptoms in this stage; viz. preliminary disturbances of the bowels. There are doubtless other preliminary signs which are sometimes felt for a few hours, or even days, by patients, before passing into the well-marked stages. Twining, Annesley, Orton, and others mention among these preliminary symptoms a feeling of malaise, oppression of epigastrium, depression of spirits, pallid, anxious, and sorrowful cast of countenance, sense of exhaustion, vertigo, noise in the ears, headache, tremor, and sense of debility. Annesley gives the following statement of the sensations of a patient, related by himself to his surgeon, Mr. Colledge: "I must knock off work, I feel unable to do more, but do not know what is the matter; I have only a little pain in the stomach and rumbling in the guts." Mr. Colledge says, sighing, peevishness, and uneasiness accompanied these complaints. Annesley relates a case in which, from the countenance of the patient, he suspected the approach of Cholera, and in which he contented himself with merely watching him. Nine hours elapsed after the establishment of his suspicions from premonitory signs, before vomiting, purging, and spasm appeared. Without doubt, after making due allowances for the fancies and terror of people during Cholera epidemics, preliminary disturbances of the nervous system have occurred, and should not be made light of. But it must be acknowledged that in the majority of cases no such forewarnings are perceived. A premonitory symptom for which medical aid is often called, during Cholera epidemics, is diarrhœa. The disease itself often begins suddenly with purging or vomiting, but in numerous cases there is relaxation of the bowels for some days or hours before the real attack begins: the motions, watery or semifluid, sometimes pale, but not always so; three, four, or more in the twenty-four hours, perhaps with griping. There may be some sense of exhaustion with this. There is a strong tendency to diarrhœal complaints during Cholera epidemics, and, though some of the cases may be harmless, many of them do ultimately pass into Cholera. The necessity of checking such discharges should be earnestly impressed upon the medical practitioner.

Stage of Development.—Evacuation Stage.—It is a matter of common observation that the attack commences with purging, very often early in the morning; vomiting seldom comes on till later. The alvine evacuations are copious and fluid. The first stools, generally, consist of the ordinary contents of the intestines mixed with much liquid. The patient often describes them as rushing from him in a full stream. Often so great is the purging, that he sits sometimes on the close stool or privy, until several pints or even quarts of fluid have passed from him. Generally the evacuations are repeated and frequent; they are soon attended with a feeling of exhaustion, so that the patient is glad to get to his bed again. This excessive watery purging characterises the onset of Cholera. It is frequently painless, but not always so; therefore we should not suppose that a patient has not Cholera because he has griping and pain. So excessive are these evacuations, that in two or three hours, or less, an ordinary-sized stool-pan will be nearly filled. With the exception of those first passed, they are of a light straw or pale drab colour. The surgeon should not be thrown off his guard because the evacuations he sees in mass are somewhat coloured. There is often fæcal matter enough in the first evacuations to colour the subsequent stools passed into the same unemptied vessel. The quantity and consistence of the discharge and the effect upon the pulse are of infinitely more importance than the shade of colour. The name of rice-water stool has been given to the genuine unmixed Cholera evacuation, and if collected separately, after the first two or three have been passed, the discharges do, indeed, resemble water in which rice has been boiled. It is a thin, pale, slightly opaque or slightly turbid fluid, depositing a sediment on standing, which is like fine, minute, flaky particles of rice broken down by long boiling. Occasionally in the evacuation stage, the fluid is whitish or somewhat milky, or of shades varying between this and the whey-like colour before mentioned. With purging, but generally beginning later than it, is combined vomiting. The fluid vomited, if unmixed with ingesta, is clear and watery, often in quantities of a pint or more, and generally ejected with force. The vomiting is less constant in its intensity than the purging, and sometimes is very slight, occurs at irregular intervals, and is readily excited by medicine or drink. When the rice-water evacuations appear, cramps generally set in, not often before this; they are most frequent in the fingers and toes, in the calves of the legs, thighs, and sometimes in the abdomen; they may continue through the next stage. By the time that the vomiting and purging have become fairly established, and even in the earlier stages, the countenance becomes altered. It assumes a somewhat leaden hue, and has a tendency to shrinking; a stony, staring look, with the capillary circulation sluggish. Alteration of countenance often points out the character of the disease, even before any symptoms have been complained of, though they may really have existed for some hours. Whatever may be the state of the countenance, if there have been many stools, or frequent vomiting, symptoms of depression appear; the pulse begins to lose its strength and soundness, and the temperature of the surface falls. Within six or seven hours from the onset of the purging, or even much earlier, the pulse may dwindle down to the faintest thread, or may entirely disappear from the wrist for many hours; in others, even in well-marked cases, though in milder forms of the disease, never quite ceasing to be felt at the wrist. This period is one of the greatest interest to the surgeon. With the utmost anxiety he watches, hour after hour, the waning, or the stationary, or the returning strength of the pulse, its maintenance or its extinction heralding either a happy issue in the first stages, or a future struggle of the deepest danger through the next stage, that of collapse. In some cases the balance oscillates for hours. If it turn adversely, with the failing circulation come the shrunken face, the lessening of bodily warmth, and the greater exhaustion; and the patient passes into the stage of collapse.

Stage of Collapse.—Algide Stage.—This is very much more dangerous than the last. Evacuations from the stomach and bowels are now less frequent and copious. The alvine discharges generally contain less liquid; sometimes still of many ounces in each, at others merely a little clear fluid with gelatinous mucous-like flakes or masses. The stomach often acts, violently, perhaps, but with less discharge. In the extreme state of the collapse the patient nearly resembles a corpse. There is the utmost depression possible with a capability of recovery. When this stage is fully formed, the patient no longer rises from his bed, though he often tosses about on it with sudden and frantic jerks, throwing off all covering, as if intensely hot, and seeking for cool air. Or he half springs up in bed shrieking from agonizing cramps, or he is still more exhausted with irregular paroxysmal gasping for breath, which subsides to be again renewed, and may end in the respiration being more constantly embarrassed. He passes stools under him, heedlessly or with indifference. Now, the features are shrunk and livid, the eyeballs small and buried in their orbits, the lower lid drooping, and the eye half open. The surface is deadly cold, except, perhaps, sometimes the forehead or præcordia, the tongue icy to the touch, the very breath a cold air stream, the temperature in the mouth 79° to 88° Fahrenheit, in the axilla 90° to 97°. The general surface is pale, bluish, or livid; often bâthed in cold profuse sweats, as may be the forehead, and the shrivelled hands and feet, the hands looking as if they had been long soaked in water; the pulse absent from the wrist, and in very bad cases from the brachial artery; the blood obtained with difficulty by venesection, sometimes not at all, and, when drawn, thickish or tar-like; the mind apathetic, so that between the times that he is roused by cramps or is involuntarily restless, the patient lies on his side or back, heedless of surrounding objects or persons, but not comatose until, perhaps, the last. Thirst and sense of heat of epigastrium are at times intense and tormenting; water! water! is the urgent and frequent supplication. If given to him, the sufferer spasmodically rises on his elbow, and gulps down the cooling liquid with intense eagerness, perhaps to vomit it up immediately. The voice is feeble, whispering, or suppressed. Muscular strength is almost always greatly reduced, but in some instances it is retained to an extraordinary and disproportionate extent. A man may be sometimes seen to walk across a ward, or to sit up in bed, or to rise to stool, whose pulse is imperceptible at the wrist. In extreme collapse the circulation is at its lowest ebb; the cardiac sounds scarcely, if at all, distinguishable; the respiration is embarrassed, or with alternations of quiet breathing and difficult paroxysms, and, in the later stages of fatal cases, always difficult; the dusky hue of the surface, the coldness and dyspnœa, all show great stagnation in the pulmonary vessels. Absorption, which in the early stages is strong enough to remove collections of fluid from the smaller or even larger shut cavities, is now nearly if not quite arrested. Glandular secretions appear to be stopped also, though at times milk seems to have formed in the breasts of nursing women. The alvine discharges are less, consisting of remnants of fluid exuded before marked collapse came on; the motions contain a little mucus with a small quantity of liquid. There may, nevertheless, be much matter retained in the intestines, the muscular coats of which are too feeble to expel it. The stomach is less frequently excited to vomiting, and there is an absence of saliva in the mouth. The changes between the atmosphere and the blood, and between the blood and the tissues, are as low as is compatible with life; the amount of carbonic acid gas thrown off from the lungs is about 1·5 per cent. instead of about 4. The evacuations are not generally of bad smell; but in some prolonged cases, in which there has been retention in the bowels, they are very offensive in the later stages; similar to rotten fish.

There is no more distressing state to witness than that of a patient in the state of collapse from Cholera. The contrast between his state of a few hours previously, the sudden affliction of friends and relations, the apparently hopeless condition of the sufferer, all conspire to impress one painfully. But with all this, hope need not be utterly cast aside. The patient may lie for hours without a pulse at the wrist, and sometimes even in the brachial artery, and yet recover. Cases without pulse in the brachial artery and manifest permanent impeded pulmonary circulation, with dusky and livid countenance, seldom or never recover; but short of this a large number do.

The state of collapse may last for hours, twelve to forty-eight, or in very rare cases even longer, and yet recovery may take place. The patient may die in three or four, or he may live twenty-four or more, the pulse never returning, the respiration becoming more and more impeded, the brain more and more torpid, with onset of coma, moaning respiration, and closing in death. Or, after lasting a variable time, five, ten, twenty hours or more, the patient becomes less restless, less thirsty, and jactitation and anxiety give way to calm; he dozes quietly with easy respiration, and this is of most favourable import. The pulse at the wrist flickers, we are in doubt if we do not feel some movement, then we are certain that we do, then this beat is unmistakeably established, the superficial veins show themselves filling at the back of the hands, the surface is less cold, the countenance assumes more and more its natural character, and even becomes flushed, respiration is quiet and regular, temperature rises, and reaction may be considered certain.

Stage of Reaction.—The patient has now passed through the collapse, and is, in many instances, nearly himself again. In some persons recovery is as rapid as decline. The secretions are readily performed, the strength returns, and health is the speedy issue. Twining mentions the rapidity with which many Cholera patients recover, and that it is not uncommon to see a man well on the third day after an attack of the worst symptoms. Mr. Grainger says, "I have seen a man stand at his door on Wednesday, who on Monday was in a perfect collapse." Such cases were common in India in Twining's day. They are not so common in Calcutta now. When the collapse has passed away, our anxiety is not at an end. The perfect establishment of the secretions is our next desire. As said before, during collapse all secretions are stopped, but after reaction has come on they should reappear. The first indication of change is in the colour and character of the alvine evacuations. Independently of the influence of drugs, such as lead, mercury, &c. we find that they become sometimes milky or whitish, then greyish, then darker or muddy, and at last brown. We find that the liver is acting, at all events as far as colouring matter is concerned. We also look anxiously for the passing of urine. We may have to wait ten, twelve, or thirty hours, or even more. At first it comes scantily, high coloured, acid, an ounce or two with a strong and peculiar animal smell, deficient in urea perhaps, but not in all animal principles, generally albuminous with many transparent casts. The albumen or allied compound when present is not always detected by nitric acid, though often by heat, giving a deposit not dissolved by nitric acid when the nitric acid test alone failed to detect it. The urine often turns pinkish with nitric acid. After the first, the secretion becomes copious, and probably by this time all the functions of the system will have become natural.

The above description applies to the well-marked evacuation and algide stages of severe ordinary cases met with in the present day in India. It is not to be supposed that all these symptoms occur in every instance, or that they are always equally well developed. There is not always the same degree of collapse, the same duration of stages, the same apparent amount of evacuations. The cases sketched above are such as have the characteristic evacuation stage, lasting six or eight hours or more; followed by decided and complete collapse. Modifications and variations will be mentioned hereafter.

As has already been said, the stage of reaction is not always the period of safety. When the algide stage does not last more than eight or ten hours, there is hope of regular convalescence; but when it goes beyond this—say eighteen or twenty-four, or more—we must, in the event of reaction, expect a much larger proportion of secondary risks.

It seems that in the earlier known epidemics in India the proportion of cases with healthy reaction and of rapid recoveries to those of consecutive disease was greater than it is in the present day. In the great epidemic of 1861, about 22 per cent. of the fatal cases died in the reaction stages. In England the opinion is prevalent that consecutive risks are less common in India than in Europe. Dr. Morehead shows that this is a mistake, and says that the practitioners in India are as familiar as those of Europe with all the diseases of the reaction stages. I think that most of the surgeons in Lower Bengal will confirm this opinion. Dr. J. Macpherson, in a paper on the Indian Annals, shows that for a period of ten years, one-fifth of the fatal cases of Cholera in the Calcutta General Hospital died after the stage of collapse was over. Whatever may have been the case formerly, I can bear testimony to the large proportion of secondary affections which are now met with in Calcutta, both among Europeans and natives. I regret that writing this paper in England, away from my own hospital records, I cannot now avail myself of them to prove this and many other points by figures. In the Report of the General Board of Health on the Cholera epidemic of 1853-54, it appears that in 1,777 deaths, 249, or about 14 per cent. died of consecutive fever. Dr. Gull states that probably not more than one-tenth died of it in 1848-49. In the North-West Provinces of India in 1861, as before said, the proportion was 22 per cent. which is larger than it was in England. I have, therefore, little doubt that in India, or at all events in Lower Bengal, the diseases following reaction are quite as frequently seen as in England. The diseases succeeding to collapse may be arranged under the following heads:—

1st.—Imperfect reaction, relapse and occasional deviations from normal convalescence.

2d.—Complicated reaction ending in uræmia, low fevers, gastro-enteritis, diarrhœas, and various asthenic sequelæ.

Relapse after the circulation has been fairly restored, is not very common, but it is met with now and then. There is a return of vomiting, purging, exhaustion, and death in a few hours or two or three days. In one case known to me it occurred after the administration of a very mild dose of a resinous purgative. Dr. Johnson mentions a case of relapse after some pears had been eaten. In the case of a lady whom I attended the evacuation and algide stages were passed safely through; during the next day great dyspnœa came on, and death, with all the symptoms of general collapse, followed. In this case I thought that the great disturbance of the respiration was due to collapse of the lung tissue.

The milder deviations from ordinary convalescence are slight febrile excitement, obstinate vomiting, hiccup, indigestion, and want of sleep.

Mild Fever.—In some of the cases after collapse the patient appears drowsy and listless, and, though generally easily moved, likes to be quiet. There is often a slight febrile movement with this, the tongue is dryish in the centre, the epithelial coating of the lips and mucous membrane of the cheeks thickened and opaque, and superficial abrasions occur opposite the most prominent teeth. The secretions appear to go on; the appetite is absent. In most instances this state does not last more than four or five days, and perfect recovery follows; or the fever assumes more or less of a remittent or intermittent character, which passes off in a few days; or sometimes it ends in coma or decided typhoid symptoms. Occasionally the fever terminates with an eruption somewhat resembling urticaria; in some cases erythema, which may become prominent all over the body, last two or three days, and then disappear with slight burning desquamation. It is not dangerous. The patient generally recovers as the eruption goes off. In some instances a red exanthematous efflorescence appears for a day or two instead of the urticarial elevations. The fever may be accompanied with irritation of the gastric mucous membrane and slight diarrhœa, but these are not essential.

Vomiting.—Irritability of the stomach may be caused by some degree of congestion of its mucous membrane approaching to subacute gastritis, owing to the frequent straining and vomiting, or to stimulants incautiously given. It is not necessarily accompanied with feverishness, but there is generally thirst and burning heat of œsophagus and at epigastrium. The patient cannot retain nourishment at first; the smallest amount being at once rejected. This condition often lasts several days, and requires great care and attention. I do not remember to have seen it fatal. When existing as the only symptom, great debility attends it, and convalescence is often delayed many days by its continuance. Sometimes it passes into a dangerous state of gastro-enteritis. Hiccup annoys the patient greatly, interfering with his rest, and is generally present with loss of appetite. It is not always constant: sometimes it leaves the patient intervals of some hours, then comes and goes again. It is generally accompanied with great eructation of gas, but is sometimes without it. It is not dangerous, though it is very annoying, and distresses the patient so much that he anxiously demands relief.

Want of Sleep.—This is very irksome when prolonged. I have known it to last for two or three days or more, the patient longing for sleep, and restless, and imploring for medicines to soothe him to repose.

Imperfect Reaction.—This is somewhat similar to relapse, but reaction does not proceed in it as in those which I have called relapse cases. In this imperfect reaction, the symptoms of great collapse pass away or diminish; the pulse improves, but does not regain its natural strength; the temperature remains low; the purging, though abated, continues, the evacuations watery, though often slightly tinged with bile; the vomiting may continue; there is anorexia and much prostration; the urine flows freely, and is apparently natural. Though time passes on, the patient does not get stronger; he lives three or four days, and then sinks from exhaustion, with disturbed respiration and coma. It may, ultimately, if the case is sufficiently prolonged, pass into the typhoid state. There is no pyrexia with this important reaction. It is not always fatal, but very frequently is so.

Uræmia.—This is very common. The symptoms of collapse pass off. If the urine is not secreted, we are not long without indications of mischief. Vomiting often returns soon, the ejecta not now colourless, but generally grass-green. Uræmia shortly succeeds. The patient has weight about the loins, listlessness, delirium, drowsiness, coma; the eyes become injected and dark, the tongue dry, the teeth foul. Before coma comes on there often exists a singular state of muscular resistance; the patient opposes all attempts at overcoming the ordinary contraction of the muscles. He strongly resists the opening of the eyelids, the depression of the lower jaw, or the straightening of the fore-arm. There is not spasm but simple resistance. Dr. Morehead notes that uræmia is preceded by preternatural slowness of pulse. If the renal secretion is not established, the coma becomes deeper and deeper, and death may occur within forty-eight hours after the full reaction; but it is generally delayed longer than this; and even after secretion is apparently restored, the patient may pass into the typhoid state. The length of the collapse has doubtless an influence upon the suspension of the renal secretion; the shorter it is,—its duration not exceeding eight or ten hours,—the greater probability is there that the kidneys will act easily within twenty-four hours. We must not despair, however, if the secretion is retarded for twenty-four or even thirty-six hours after reaction. In extreme cases even this limit may be passed. Dr. Morehead notices that, when the collapse has lasted eighteen hours or more, there is much greater danger of consecutive disease depending on defective secretion; and the more speedily the full circulation has been restored after long collapse, the greater is the risk from continued suppressed urinary secretion. The comatose cases are very frequently fatal, but not necessarily so. If the kidneys act, bad symptoms will probably diminish; but if they do not, death is inevitable. The secretion of urine, however, does not always relieve the head symptoms, as Dr. Morehead has also pointed out. The bowels are sometimes relaxed, sometimes constipated; the evacuations yellow and feculent. Diarrhœa may carry off some urea, and should not be checked. The whole train of symptoms of uræmia need not be described here. They are the same as in uræmia generally. There is no febrile action, or it is but slight.

Cholera Typhoid.—Writers do not always distinguish between this and uræmia. Uræmia may pass into a typhoid state, but there does exist a consecutive fever independently of uræmia; or at any rate without suppression of urine, or without albuminous urine. Possibly the blood may not be fully purified of urea, and a quantitative investigation might prove a deficiency of excretion, but in some of the cases I have found sufficient urea to crystallize freely on a glass microscope slide. I have never made a quantitative examination for the twenty-four hours. It is likely that other principles, besides urea, indicative of the blood being loaded with effete matter, might be discovered, as in the cases mentioned by Dr. Letheby. In cases of consecutive fever, the patient for two or three days after the collapse may appear to be doing fairly, the excretions and secretions appear to go on sufficiently; then the tongue becomes dryish, and a little quickening of the pulse comes on during some part of the twenty-four hours; perhaps towards evening. Often the febrile movements are pretty regularly paroxysmal. The excitement is not high, and the pulse, though quicker, is generally weak and small. In a short time the patient passes into a typhoid condition; sometimes passing through all the risks of the extreme adynamic state; very often dying; but the disease is not necessarily fatal. The duration of the fever varies. I have known a patient to die twenty days after the collapse had passed away, the urinary and hepatic secretions being apparently natural throughout. Probably the majority die about the eighth to twelfth day. This fever may possess all the usual symptoms of adynamic fevers—low delirium, coma, dry tongue, parched lips, feeble and quick pulse, bed-sores, liquefaction of the blood, purpura, &c. It would be superfluous to describe more fully all the signs of the typhoid state. The patient may sink under it, or slowly recover, and remain for a long time in an anæmic, debilitated condition. Coma sometimes comes on within the first day or two after reaction. In some cases this may depend upon sudden poisoning by urea, but in many the secretions do not seem to have been in fault. It is generally fatal.

Ulceration of the Cornea.—Disintegration of the cornea is sometimes a sequel of Cholera in India: most frequently among natives, but I have seen it in Europeans also. It comes on a few days after the reaction has set in. An asthenic state of the system is necessary to it. Generally there are some typhoid symptoms, but it does not always appear in the worst state of the low fevers. The lower segment of the cornea is the part to suffer first. It is not caused by inflammation; in the early stages, not a trace of an enlarged vessel may be visible. During the worst part of the collapse the lower eyelid falls away from the upper, the lower segment of the cornea is exposed, and the uncovered part of the epithelial layer at the time often looks dryish and parchment-like. It is probable that this leads to the subsequent disintegration of the part; but whether this be so or not, the essential cause of the affection is defective nutrition of the cornea, as in the case of Majendie's dogs. We find that in four or five days, or later, after reaction a part of the lower segment of the cornea on one side becomes hazy; the area of cloudiness not very accurately defined. There is so far no discoverable disintegration. In twenty-four hours the epithelial layer over part of the opaque area shows abrasion, or there may be some loss of substance of the deeper layers of the cornea, producing a minute curved groove about one twenty-fourth of an inch from the corneal margin; the groove is less than the hazy part. By this time the opposite eye has generally become hazy in the same manner as the first had been on the previous day. In twenty-four hours more the groove in the first eye may have doubled its size. Examined with a lens, the surface of the groove is of a dirty ash colour, and opaque, having indeed all the character of a minute slough. The ulcer may spread, and penetrate the entire thickness of the cornea, and extend at its margins, but in the majority of cases the patient dies before such extensive destruction occurs. The mischief is generally symmetrical; but the eye last attacked is less advanced than the first. If the patient recovers, the eye is generally preserved without any very serious disfigurement. With his general improvement the ulcers also improve. There is first arrest of disintegration, then diminution of haziness and opacity, then cleaning of the surface of the ulcer, which becomes beautifully clear and transparent. Cicatrization follows, perhaps rapidly, and the repair is generally perfect, so that unless the ulcer is very deep, in a few days it is often difficult to discover where the mischief has been. Permanent opacity, hernia of the iris, or staphyloma, are rarer than would be supposed. In the later stages, there may be vascularity of conjunctiva and sclerotic, but they are effects, not causes, of the ulcers. This state of the cornea is frequently met with in India, in cases of chronic diarrhœa and other debilitating diseases, and of course is not peculiar to Cholera.

Gangrene of various parts.—Among some of the rarer sequelæ, I have seen, among the natives of Bengal, complete gangrene of the penis and scrotum, and partial gangrene of the scrotum and of the point of the nose; also superficial gangrene of the mucous membrane of the mouth. I have never seen mortification of the intestines. When the mortified parts are small in extent they may be cast off, but in the extensive gangrenes of the penis and scrotum I believe that death always happens.

Parotid Glands.—Swelling and inflammation of these are not unfrequent; they often, though not always, end in suppuration. It is unnecessary to give an account of the symptoms of this affection. It appears during the second or third week, and with low fever. Both sides generally suffer in succession. The patients often sink during its progress. It is always a grave symptom, indicating a very asthenic state of the system, and interfering much with the patient's power of taking food.

Bed-sores, Boils, Ulcers, Low Inflammation of Lungs, Pleura, &c.—The patient often suffers very much from these. They often greatly retard convalescence, but do not require any special notice.

Consideration of special Symptoms.—It may be well now to consider more in detail some points not fully spoken of in the foregoing account of the symptoms, in order not to interrupt their description. The intestinal discharges require more notice. The quantity evacuated is generally considerable. It is difficult to ascertain this in hospital patients. They amount frequently to 80, 100, or 150 ounces during the evacuation stages. After the feculent portions of the stools have passed they have not much smell; but in the later stages, as before mentioned, if they have been retained in the intestines, they are often very offensive, but they are not so always. The composition of the Cholera stool has been studied chemically and microscopically. The Cholera stool and the contents of the intestines after death are not exactly alike under microscopic examination, and Dr. Parkes has pointed out the necessity of remembering that Boehm's observations are applicable to the intestinal contents, and not to the discharges collected during life. As before said, the Cholera stool separates on standing into a thin whey-like fluid, and a variable quantity of sediment. The specific gravity of the liquid portion is 1,005 to 1,010. The sedimentary flakes and particles, according to Parkes, Gull, Gairdner, and others, contain organic forms, which are described as follows, lying in a hyaline basis :—

1. Amorphous granular matter and larger granules, often very abundant.
2. Minute bodies having the general character of nuclei, $\frac{1}{250}$ to $\frac{1}{180}$ line in diameter.
3. Fine granular cells; some large, some resembling pus cells.
4. A very small quantity of scaly epithelium, generally not easily discernible.

Dr. Parkes states that the actual quantity of sediment is very small; the dried deposit from a pint by measure of a Cholera stool weighed four grains only. The composition of the solid or gelatinous matter of the stools is doubtful: Parkes considers it some modification of fibrine; Gairdner thinks that it presents the reactions of mucus. It is possibly some modification of mucus.

In contrast with the above, the microscopic appearances of the contents of the intestines found after death, as first described by Boehm, consist of a large quantity of epithelium in various states of aggregation, separate cells, and flakes which are coherent masses of greater or less extent, comprising the coverings of small regions, as apices of villi, or covering the whole villi and their bases in variable quantities. According to the state of aggregation or distinctness of the epithelial particles, and the quantity of fluid in which they are mixed, the appearance of the whole may vary from a rice-water or milky to a creamy, purulent, flocculent, or oatmeal consistence. Dr. Parkes has shown that there are, mixed with these, other organic forms found in the stools. There is very little epithelium found in the contents of the colon. The great difference in the microscopic appearances of the Cholera stools and the contents of the small intestines after death, is in the nearly complete absence of epithelial particles in the stools. The inference from this and other considerations is, that the shedding of the epithelium does not take place during life, but that it is due to post-mortem maceration and detachment.

Chemistry of the Evacuation.—The specific gravity of the liquid part is seldom above 1,012 in the height of the stage of discharge. The reaction is faintly alkaline or neutral. Dr. Parkes, in the February number of the "London Journal of Medicine," for 1849, has given us valuable information as to the composition of the stools, and shown that the liquid portion is not similar to the serum of the blood; that it contains but little albumen, and consists chiefly of the water of the blood with saline matter and a small quantity of animal matter. The following table, copied from his papers, shows at a glance the real character of the discharges.

Period of the Disease in which the Stool was passed.	Specific Gravity.	Albumen in 1,000 parts.	Extractive in 1,000 parts.	Sol. Salts in 1,000 parts.	Total of Solids in 1,000 parts.
Diarrhœal period	1,012·9	0·466	3·846	9·04	13·9
Diarrhœal period	—	0·29	6·82	5·99	13·1
Early Algide stage	1,009	2·4	1·27	10·98	14·65
Developed and Intense Algide stage	1,009·5	1·18	0·55	9·14	10·87
Developed and Intense Algide stage	—	2·186		7·52	9·706
Developed and Moderate Algide stage	1,008·3	0·27	2·23	8·33	10·83
Developed and Moderate Algide stage	1,005·8	3·2		5·827	8·947
Commencement of reaction	1,014·0	20·84		6·34	27·18
Commencement of reaction	1,008·91	1·48	6·055	9·085	16·62
Relapse	1,017,83	0·855	17·355		18·21
Relapse	—	not weighable.	4·589	3·881	8·47

Nitric acid occasionally gives red reaction in the liquid. This is probably due to a small quantity of bile mixed somehow with the evacuations, but it is not certain what it is.

Blood is rare, but it has occasionally been met with both in the stools and in the contents of the intestines. The pink or reddish discharges often consist only of the colouring matter of the blood, the red corpuscles being generally absent.

Although the albumen and animal matter found in the stools is very small, the quantity of salts contained is considerable. The greatest quantitative loss which the blood suffers is in its watery element. For every 100 ounces passed in the fluid evacuation stage, the loss to the blood in water is 98 to 99 ounces, and of salts nearly or about one ounce. The salts exuded are the chlorides of sodium and potassium, phosphate of soda, carbonate and sulphate of soda, bearing a proportion of seven or eight parts in 1,000; a proportion nearly resembling the quantity in the blood within the vessels. The earthy phosphates do not pass through the mucous membrane as in health. In three analyses by Dr. D. Thompson a larger proportion of organic matter was found, but Dr. Parkes's observations probably hold good for the majority of cases. It will be seen that the intestinal surface removes from the blood a large quantity of water, a small quantity of animal matter, and much saline matter, doubtless causing great change in the blood, and in the behaviour of the different elements of the blood to each other. The chemistry of the vomited matters has been less studied. In six cases examined by Becquerel, the solid matter in 1,000 parts varied from 6·37 to 54·70 per 1,000, the albumen from a non-weighable quantity to 31·50 per 1,000, the chloride of sodium from 2·35 to 8·24 per 1,000. No mention is made of the matters taken into the stomach, nor the period of the disease in which the discharges were collected.

Blood.—The viscidity of the blood in the algide stage has been mentioned. Chemistry shows that it has undergone considerable changes. The analyses most known are those by Sir W. O'Shaughnessy, Drs. Parkes, Garrod, and Schmidt. They show a diminution of water and a relatively increased proportion of solids. According to Garrod's, taking the maximum of solids in health for males at 240, and for females at 227 per 1,000, he found that the total solids in seven cases were 251, 360, 271, 271, 275, 282 and 284. The blood-globules and albumen were increased; the alteration in quantity of the fibrine was doubtful, but it was less coagulable, and it was probably altered in quality. Dr. Garrod's observations were made on blood obtained, after death, from the cavities of the heart and neighbouring great vessels. Dr. Garrod and Dr. O'Shaughnessy differ as to the proportion of salts. The former thought that they bore as high, if not a higher proportion than in health; the latter that they were diminished. Dr. Schmidt's observations were made on blood obtained by venesection. His results show an increased density of the blood and of the morphological elements in proportion to the duration of the exudation process from the surface of the intestinal canal; a relative increase of the solids in the blood, so that after thirty-six hours of the exudation process they reach to nearly half more than their normal proportion. Schmidt thought that the inorganic salts were diminished in the later stages of the exudation process, so that the proportion of organic matter in the serum was doubled. The specific gravity of the fluid is higher than natural; 1,076 to 1,081 instead of 1,062 to 1,060. The blood is sometimes found acid. Dr. Garrod thinks that this is due to the impeded excretion of organic acids. Urea in small quantities is sometimes found in the collapse, and generally in the reaction stages. In the main, all the analyses support each other, showing in the collapse stage a greater consistence of the blood, higher proportion of organic solids of all kinds, with impaired coagulability of fibrine, higher specific gravity, occasional acidity, occasional presence of urea, and an undecided proportion of inorganic matter, but nevertheless, probably, a diminution in the absolute quantity of saline elements. In the reaction stages they show often a considerable quantity of urea and softness of the coagula.

VARIETIES.—As before said, the description of the symptoms given applies to the ordinary cases of Cholera, but we occasionally meet with instances which in some points differ from them. Some of the cases run a much more rapid course; thus in some the evacuation stages are shorter, lasting two or three hours only, and collapse may come on with or without much discharge from the stomach and bowels. Others appear yet more rapidly fatal. Thus at Kurrachee, in 1846, people are said to have died within less than an hour from the time that they were seized. Dr. Milroy states that at Teheran, in 1846, those who were attacked dropped down suddenly in a state of lethargy, and died at the end of two or three hours without convulsions or vomiting, but from a complete stagnation of blood. In these cases it is said that the dose of poison is so strong that death occurs before there has been time for exudation to take place. Mr. Thom says: "Among the first hundred cases which occurred many died in a few hours, and some in less time; one man I am told went off in less than an hour. In these vomiting and purging were not always present. Sudden collapse, ending in profuse sweating, were the most prominent symptoms—in fact asphyxia had already taken place. It was often found that the pulse had ceased at the wrist, the eyes turned up, the voice hollow and feeble, before the natural hue had given way to that horrible lividity which is characteristic of the disease, so instantaneously was the power of life arrested." And again, "The next class of cases were those in which the first seizure was equally sudden, and the collapse preceded the vomiting and purging. There were sudden faintness, prostration of strength, restlessness and anxiety, accompanied by vertigo, deafness, loss of vision, alteration or hollowness of voice, weak and slow respiration performed convulsively or in sighs. These were followed by nausea, vomiting, and purging of congel-like stools, sensation of burning heat at the præcordium, intense thirst and desire for something cool," &c.

From such cases as these it has been argued that exudation from the blood is not a necessary part of Cholera. It should be recollected, however, that the amount of discharge voided is no positive measure of the amount of fluid separated from the blood during life, because it often happens that a very large amount is retained within the intestines during life. This will be again referred to.

In some of the cases there is not the same shrivelling of the body as described in the typical cases. The body retains much of its plumpness, and the skin is dry. Collapse, nevertheless, comes on early in these cases.

In a few cases the skin is warm and the perspiration warm, even during the earlier part of the collapse, which comes on early and is complete.

Mr. Twining and others describe cases which appear to have in the first stages more of a *sthenic character* than those usually met with now, and to which the name of Spasmodic Cholera was, perhaps, more applicable than to those described above. They were cases in which the "actions of the constitution were evidently febrile, and in the febrile stage of the disease were attended with violent and painful spasms, warmth of surface, and free circulation." Dr. Morehead does not find these common in Western India.

Dr. K. Mackinnon relates *another modification* of symptoms which he witnessed in a dreadful visitation of Cholera in the Tirhool jail. He says: "After a check had been put to the vomiting and purging, the voice, breathing, and warmth of skin became natural, the face had none of the peculiar character of the disease, the patients walked about and called for food, saying that they felt well; on feeling the pulse in those cases it was barely perceptible, I think in some cases not to be felt at all. In this extraordinary condition some of the men lived for two days; they all died, and, if my memory serves me, invariably by coma." Dr. Mackinnon does not allude to the state of the secretions. But the feebleness or absence of the pulse with such a degree of muscular strength and general well-doing is remarkable. I have seen some such cases, and they are mentioned by Scot and others.

Dr. John Macpherson mentions two cases with hæmatemesis, occurring in the reaction stage, and in one of them associated with hæmorrhage from the bowels.

Choleraic Diarrhœa.—This, though perhaps not a variety of Cholera, approaches that disease so nearly that it may be well to allude to it in this place. It almost always prevails during Cholera epidemics, and sometimes precedes it in a district for weeks or months. It is often difficult to distinguish from Cholera at its onset, and into this, indeed, it often passes. Some of the cases have so much the appearance of mild attacks of Cholera that it is hardly possible to draw a line between them and the real disease. At Paris the name of Cholerine was given to these diarrhœas. It is probable that the diarrhœa is due to a milder dose of the poison than suffices to produce true Cholera. The patient's most prominent symptoms are those of diarrhœa. This begins in the night or early in the morning, and without any assignable cause. The motions are three or four, or sometimes six or seven in twenty-four hours—passed without effort or straining or griping. This diarrhœa is generally painless, which circumstance, combined perhaps with an apathetic state of the mind, is the cause of the disease being frequently allowed to run on unheeded. The evacuations are more or less feculent, but very liquid, and generally copious; the colour varies from brown to light yellow or pale straw, and when the disease passes into Cholera the motions are increased, and assume the rice-water character. There may be vomiting, and sometimes cramp. There is generally a good deal of sense of exhaustion and weakness. The patient has not much appetite, and is more or less ill. The diarrhœa may last twelve or fifteen days, but may at any time pass into Cholera. The symptoms appear to be amenable to treatment, but the disease may destroy life by exhaustion without passing into Cholera or collapse. It is difficult to estimate the mortality of this affection, because so many of the cases are set down as simple diarrhœa, and when they pass into Cholera they are recorded as Cholera. The mortality is not great, however, of such as remain classified as diarrhœa. The deaths from diarrhœa in the Cholera season of 1854 appeared to vary from 6 to 18 per 1,000. The great success in the treatment of these shows how desirable it is to check all cases of Choleraic diarrhœa; by so doing, there is little doubt that many cases of Cholera are arrested.

Cholera Fever.—It has been frequently noticed that at certain periods of the Cholera epidemic, towards its decline, the Choleraic diarrhœas pass into a sort of low fever. In speaking of the Kurrachee epidemic, Mr. Thom says, as Cholera closed its career it gradually changed its type to that of fever; while one out of four or five cases ran into Spasmodic Cholera, the others would terminate in fever, and were registered as such, showing all the characters of a low remittent. Sir Ranald Martin speaks of "Cholera Fever" occurring in Calcutta in 1834, and at other times during the Cholera season, which was remarkable for diarrhœa, and for the tendency which this had to run into Cholera, especially under doses of saline and drastic purgatives, and when these were administered over night. It is not so easy to say what is the connexion between these fevers and Cholera. In Calcutta one frequently meets with fevers which begin with diarrhœa. I have elsewhere mentioned this as frequently ushering in the red fever sometimes prevailing there, and it is not unusual for some of the common fevers of children to begin in this way. It is probable that some of these happening to prevail during Cholera times, are influenced by the epidemic constitution of the atmosphere. It is not likely that the fever is a variety of Cholera. We should learn from them the practical lesson of not giving much purgative medicine, even in fever, during Cholera seasons, and especially to avoid salines and hydrogogue purgatives, and the administration of any at bedtime likely to operate in the course of the night or morning.

In classifying cases of Cholera, we may, excluding Choleraic diarrhœas and fevers with Choleraic diarrhœa, arrange them as follow:—

1. Those in which collapse is not perfect.
2. Those which after vomiting and purging pass into well-marked and complete collapse.
3. Those in which collapse comes on with little or no apparent evacuation.
4. The sthenic cases.

The cases most frequently met with are of the second class.

DURATION.—This may, including sequelæ, vary from two to three hours in the worst form, to several weeks in those protracted by secondary disease. The time which elapses from the first symptoms of choleraic evacuations to the period of reaction is more limited. In the Report of the Board of Health on the epidemic of 1853-4, the duration of 1,744 fatal cases from the first symptoms is stated to have been an average of not less than 64 hours; the duration of 1,856 cases of recovery, 9·06 days. In the general registry of deaths, it is mentioned in the same Report, 9,590 fatal cases lasted an average of 2·39 days.

The duration of the stages has been already alluded to; the diarrhœal may scarcely exist (excepting Choleraic diarrhœa), or extend to 24 hours; the collapse stage may terminate fatally in two hours, or it may be prolonged in extreme cases to 70 or 80; the reaction stages may terminate favourably within 48 hours, or may linger on for many weeks, when complicated with fever or other sequelæ.

MORTALITY OF THE DISEASE.—This is very large, and the averages approach each other in different climates and countries. The mortality to strength of a population may vary greatly, but it has been observed for England, that, whether in healthy or unhealthy districts, the deaths to cases are pretty even; the difference in mortality between such places depending more upon insusceptibility of the population in healthy places than on any actual difference of violence of the disease. In India the deaths to cases do seem to vary in different ranks of the army: the officers having a lower mortality than the men. We may say in round numbers that more than 50 per cent. of the attacked recover. In some epidemics the mortality is higher, but in others, lower: in some, 70 to 80 per cent.; in others 20 to 30 per cent. The mortality varies also with the period of the epidemic. It is generally much higher in the beginning than in the end or towards the end. It varies also in the different stages, being greatest in the collapse stage.

In illustration of the above, we find that in 1832, in England, the deaths to cases were 47 per cent.; in 1848–49, 45 per cent.; and in 1853–54, 46 per cent. In Dr. Ewart's tables for European soldiers in India, for eight years ending 1853-4, it was: for privates, 40·74; officers, 16·66; women, 31·74; and children, 39·163 per cent. For the native soldiery in the three presidencies, for various long periods of years: for Bengal, 30·54; Bombay, 33·06; Madras, 42·91 per cent. In the Report of the Cholera Commission for the epidemic of 1861, we find that the mortality of the European soldiery for that season was very high all over the provinces; the average being 63·8 for all classes in the regiments, but it varied from a minimum of 42·6 per cent. to a maximum of 80 per cent. among bodies of men in cantonments, of strength not below 500. The mortality to cases among the native Indian populations is not ascertainable. For native prisoners in jails for twenty-one years, ending 1853, it was 42·6 per cent. With reference to the excess of mortality in the beginning of epidemics, we find that in 1854, in England, in the fortnight from July 16th to 29th, it varied from a maximum of 59 per cent. to a minimum of 40 per cent.; but in the two last fortnights ending November 16th they were only 23·21 per cent. In the severe outburst at Kurrachee, almost all the cases were fatal, and after that they became much more amenable to treatment. Indeed this is the experience furnished by nearly all the epidemics. The greater mortality in the collapse stage may be seen in the results of the Indian epidemic of 1861, in which we find that of 927 deaths, 717, or 77·4 per cent. died in the collapse stage.

An opinion prevails that Cholera is becoming more fatal to European troops in India than it was formerly. Dr. Ewart's tables seem to show, that though the disease is less frequent than it used to be, the ratio of deaths to cases has largely increased. Thus, in Bengal, for eighteen years, from 1818 to 1835 inclusive, the percentage of admissions to strength was 3·25, and the deaths to admissions 26·36, or 263 deaths per 1,000 attacks; while for the eighteen years from 1836 to 1853-54 inclusive, the admissions to strength were 2·62, and the deaths to admissions 39·75, or 397 deaths in 1,000 attacks; showing an average increase of mortality to attacks of above 13 per cent. or at the rate of 134 per 1,000 above the rate of the first series of eighteen years. In Madras, from 1829 to 1838, the deaths were 271 per 1,000 cases, and from 1842 to 1851-52, 502 per 1,000; or an increase of 231 deaths per 1,000 cases. The increase has taken place through all the presidencies, but in Madras it seems to have been the greatest. It is to be hoped that this increase is not permanent, and that the averages may again descend. In another of Dr. Ewart's tables, the time is divided into quinquennial periods, and it appears that although, in the main, there has been a rise in the mortality within the last and in the later quinquennial period, there were considerable fluctuations in the earlier terms, descending as much as 10 or 12 per cent. in Bengal and Bombay, and then ascending. In Madras the fluctuations have not been felt, but the increase has been maintained during four quinquennial periods. It is possible that a great deal of the apparent difference has arisen from a different method of recording cases. Of late years those cases only in which the presence of the disease was decided, have been entered under the head of Cholera; and cases which would formerly have been classed under this, are now placed in the columns for Diarrhœa. On turning to Dr. Ewart's Table 29, it will be found that there is an increase of cases of diarrhœa in all the presidencies. The returns are not exactly for the same years as those for Cholera, but include them. Thus, in Bengal the returns from 1812 to 1832 give 11·55; and 1833 to 1853-54, 12·15 per cent. admissions to strength; in Bombay, 1803-4 to 1827-28, 6·85 per cent.; and 1828-29 to 1852-53, 12·97 per cent.; and in Madras, from 1829 to 1838, 7·8 per cent.; and 1842 to 1851, 10·3 per cent. It will be seen that in Bombay and Madras, in which the rise in mortality of Cholera is apparently the highest, the increase of percentage of admissions of diarrhœa is also the largest of the three presidencies. It is probable, as supposed by Dr. Ewart, that the diarrhœas have encroached upon the dysenteries, but I think that one may fairly claim a good share of them for cases which would have been called Cholera in the older returns. It would be useful to compare the present with the past mortality of all intestinal fluxes.

Mortality to age, sex, population, &c. has been noticed in the section on Etiology.

Diagnosis.—This is easy in marked cases. Purging, vomiting, anxious countenance, cramps, quick advent of collapse, and profuse sweating, are decisive enough of the presence of Cholera.

There is some difficulty in deciding in the diarrhœal stage. Cholera may be mistaken for bilious diarrhœa, choleraic diarrhœa, purging, produced by irritating drugs or poisons, and the onset of some fevers. We rely for diagnosis chiefly on the absence of any known cause for purging, the absence or slightness of pain or tormina, upon the liquidity and pale colour of the stools, and copious rush of fluid from the bowels; the early tendency to exhaustion, and the frequent beginning in the early morning. If the patient is passing brown feculent matter, not very liquid, and if there be griping and colic, and a probable cause in improper diet, we may be pretty easy, and the disease will probably be common diarrhœa. But we should nevertheless watch the patient carefully for a few hours. Choleraic diarrhœa is less violent in its onset than Cholera, and the stools generally contain bile, even after the disease has lasted for days. There is seldom much or any pain in this. It should always be treated as Cholera in the beginning. Vomiting helps in the diagnosis, but it is sometimes absent for the first few hours, and is sometimes present in diarrhœa. The vomiting in Cholera is fluid and colourless generally; that of bilious diarrhœa often consists of undigested food or biliary matter.

The diarrhœa in the beginning of fevers is often very exhausting, and at first it may be difficult to distinguish it from the commencement of Cholera. Generally, however, there is less exhaustion; and though there may be a tendency to collapse, this is not deep, and in a few hours the pyrexial stage comes on.

All surgeons in charge of hospitals must be familiar with indications afforded by the countenances of patients even in the earlier stages. Men are often seen looking somewhat anxious, with the face pointed, and perhaps somewhat shrunk and mottled or leaden-coloured. On being questioned, it will perhaps be found that the patient has watery purging on him; a closed stool at his bed-side will, perhaps, be half or two-thirds full of the brownish or pale liquid, and he will be, indeed, in the first stage of Cholera, though ignorant of it, and making no complaint. A glance at a man's face will often save his life, and no man, when in or out of hospital, whose countenance shows this change, should be passed over in a routine way, however trivial his own complaint may be, or although he may be engaged, as sometimes happens, in his usual occupations.

Cases of irritating poisoning often occur either from accident or design, and it is always well to be alive to such facts when called to cases supposed to be Cholera, and not take it for granted that all cases of vomiting and purging are those of Cholera.

It cannot be too strongly impressed upon the young medical practitioner that in every case of diarrhœa which he attends, in seasons of Cholera, he should ask to see the evacuations at the very first visit. In India fortunately there are few privies or cesspools to private houses, so that this inquiry is not so difficult as it might be in England. If the stools have been taken away, the next ones should be reserved. This is most important for diagnosis, and for the satisfaction of the medical man's own conscience it should never be omitted. Neither rank, nor sex, nor age of the patient should be a cause of the neglect of this.

It should be remembered that many patients brought into hospital with low adynamic fever are sufferers from the sequelæ of Cholera, and that inquiries should be made as to the mode of the beginning of the disease for satisfactory diagnosis, if not for treatment.

PATHOLOGY.—*Morbid Anatomy.*—This must be considered after death in the collapse and in the reaction stage.

(*a*) *In Collapse.*—The surface is generally bluish or mottled, especially in the dependent parts, and there are often sub-conjunctival ecchymoses. The extremities are shrivelled. The temperature, so low during life, often rises a little after death, and the body cools slowly. According to Dr. Gull, Mr. Barlow and others have noticed a rise of two or three degrees of Fahrenheit above the temperature observed just before death. Rigor mortis comes on quickly, and lasts quite as long as in other diseases. Muscular contractions have been sometimes observed, and have in rare cases been sufficiently strong to alter the position of a limb. They may come on within the first few minutes after death, and last two or three hours. Putrefactive changes are not hastened in Europe. In India they are not more rapid than in other diseases. The abdomen often feels doughy and dull on percussion, except in the upper parts.

Blood—Organs of Circulation and Respiration.—When death occurs in collapse, the disease has not lasted long enough to cause organic changes. Such changes as there are, are chiefly in the distribution of the blood. Ecchymoses are frequent. They are met with under the serous membranes, as the pleura and the pericardium; under the mucous membranes of the small and great intestines, around the dura mater of the spinal cord, and occasionally in other places.

The capillaries of the surface are empty, and the blood is chiefly found in the large vessels of the lungs and right side of the heart, and in the veins and various capillaries of the intestines. Dr. Parkes was the first to point out, in his work on Asiatic Cholera, the real seats of accumulation of blood. Most persons who have had opportunities of making post-mortem examinations of Cholera cases will confirm his statements. It was previously supposed that the lungs and heart were gorged with blood, but he pointed out accurately what was the precise situation of the congestion. He showed that the gorged parts were the vessels of the right side of the heart and the pulmonary artery in the roots of the lungs, from the right side of the heart to the smaller branches; and that the smaller vessels, the pulmonary capillaries, the pulmonary veins, and the left side of the heart, were nearly empty: in fact, that the blood was not arrested in the capillaries of the lungs as in common asphyxia, but in the arteries short of them. On section there was free bleeding from the roots of the lungs, but there was little or none in the peripheral parts; they were generally ex-sanguine. He says: "On cutting through the roots of the lungs a quantity of blood usually escaped from the divided vessels, and particularly from the *pulmonary artery*; in one case the quantity of this was two pints, in one case one pint, in one twenty-four ounces, in one three ounces, in one six ounces, and in nine between two and five ounces. In fourteen cases the lungs were completely collapsed, appearing in some cases like the lung of a fœtus. In three cases they were considerably, in eight slightly, collapsed; and in the remaining fourteen cases the collapse was in some cases altogether and in some partially prevented by old adhesions." The absence of blood in the tissue or substance of the lung was generally pretty complete, though in some instances there was more redness in the periphery than in others. The small amount of blood in the lungs seems proved by their deficient weight. Thus, he found that the average weight of the right lung in twenty-two Europeans was fourteen ounces, and of the left twelve ounces six drachms. Dr. Clendinning gives the average weight of both lungs at forty-six ounces, and Dr. Reid at forty-three ounces, showing a deficiency in the Cholera cases of fifteen to nineteen ounces. The lung tissue was flabby, with little crepitation when not collapsed, so that it may be considered to have been in an extremely anæmic state, owing to an arrest of blood before it had reached the capillaries and smaller arterial twigs. The colour of the lung on section was pale or dark, or an admixture of both. Some writers report a congested state of the posterior parts of the lungs; probably from hypostatic congestion. The right side of the heart was generally full and distended, the blood coagulated, but not firmly. Parkes found the blood incoagulable in rather more than one-fourth of his cases. The left side of the heart was nearly empty, or containing only a little black blood with a few loose coagula. In following out the situations of congestions we find that the larger hepatic veins and the branches of the portal vein, generally those leading from the stomach, duodenum, and small intestines and their small ramifications, are full both on the mucous and peritoneal surfaces of these organs. The blood is generally of dark colour, but turns brighter on exposure to the air, or to the action of the matter in the small intestines. The chemistry of the blood has already been alluded to. There is some difference of opinion as to the state of the coagulability of the blood. Parkes, Virchow, Briquet, and Mignot have noticed either that it coagulates imperfectly, or that there is a loose state of the coagulum in the great vessels. I think that I have generally observed the same. Dr. Gairdner, however, says that the coagulation in the vessels takes place much as in other diseases.

In the lungs themselves there is little change beyond those due to distribution of the blood and the collapse of the lung tissue. The muscular tissue of the heart is generally found rigid or firm.

Abdomen.—The visceral peritoneum is generally much congested; the venous capillaries darkish, becoming bright after exposure to the air; the surface duller than natural, and sometimes bedewed with a slimy moisture.

Stomach and Intestines.—Mucous membrane sometimes congested. Mucous membrane of small intestines often much injected with fine arborescent vascularity. There is little organic change in the coats; the mucous membrane may seem œdematous, and the folds rendered prominent. Boehm pointed out the prominence of the glandular structure of the mucous membrane of the small intestines, and notably of the lower part of the ileum. Thus, Peyer's patches and the solitary glands are often enlarged, the solitary more decidedly than the agminated glands. Ulceration not observed in simple cases. Occasionally patches of a slight greyish exudation have been noticed. These have been called diphtherial. If the exudation in throat diphtheria is to be taken as the pattern, I should hardly call the name a good one. These patches are not easily removed, as the diphtherial layer is from the throat. The exudation in Cholera seems more like the granular patches which are sometimes seen in the lower three or four inches of the ileum in severe cases of dysentery, and in a more highly developed form in the drab-coloured granular patches in the colon in dysentery, and which cannot be removed without some disintegration of the superficial part of the membrane on which they lie. This appearance is rare in Cholera. In many cases of Cholera, however, there is little or no congestion or decided morbid change discoverable on examination of the mucous membrane or glandular structure. The contents of the intestines have already been described. They may fill the intestines completely, or there may be but a small quantity, the consistence varying from that of an ordinary Cholera stool to a creamy or pasty, gritty-looking, greyish mass, or fluid mixed with gelatinous or fibrinous lumps or flakes, more or less adhering to the intestines. There is sometimes, but not often, a tinge of yellow, and sometimes, though rarely, a large quantity of grumous blood without any kind of ulceration being traceable.

Colon.—In the majority of cases an absence of injection of the vessels. In a small number there is venous congestion, seldom general, but in patches. The mucous membrane is not so œdematous as that of the smaller intestines, though it is sometimes somewhat swollen. There is not much epithelium mixed up with the contents. In some rare cases large ecchymoses leading to gangrene of the mucous membrane have been seen.

Liver.—Great venous trunks generally full of viscid blood. The parenchyma pale and flaccid, and often smaller than natural, with some but not constant wrinkling of the capsule. There is not often general congestion of the organ. Microscopic appearances normal. Gall-bladder generally full of bile.

Spleen generally smaller and more flaccid than natural. Some writers have described both the liver and the spleen as being congested, but this does not seem to me by any means frequently the case.

Pancreas mostly natural. The mesenteric glands are often enlarged or pale.

Kidneys.—These are generally congested, the veins, especially of the papillæ, extending outwards into the cortical substance, which is enlarged. The epithelium is generally natural in shape, very granular, probably from some protein deposit.

Brain and Membranes.—Nothing special or different from the condition in many other diseases in which the brain is not directly involved.

In summing up the post-mortem appearances in collapse, we find an alteration in the consistence of the blood; and that some of the great internal veins and the pulmonary artery are loaded with it; a great deficiency of blood in the capillaries generally except in the majority of cases in those of the intestines and kidneys; generally some prominence of the glandular structures of the intestines, and post-mortem displacement of the epithelium of the small intestines, and in many instances collapse of the lung tissue.

(*b*) *In Reaction Stages.*—The morbid appearances vary with the nature of the secondary causes of death. It will not be necessary to give a full description of these changes. In the cases of death in imperfect reaction they do not vary much from those found in collapse, except perhaps that there may be more general congestion of the lungs, more decided prominence of the glandular structures of the small intestines, and injection of the intestinal mucous membrane.

In persons dying of some of the consecutive febrile conditions, the intestines and stomach are often found vividly injected, the mucous membrane softened. The colon sometimes participates in this, and the mucous membrane is sometimes œdematous and thrown into folds, and the so-called diphtheritic exudations are met with. In some of the cases not marked with symptoms referable to the alimentary canal there is less congestion and œdema of the small intestines, as has been noticed by Reinhardt and Leubuscher.

In the uræmic cases the kidneys, when death appears early, are full and large, of purplish colour, dripping blood on section; in the later periods pale, perhaps œdematous, infiltrated with granular matter of pale colour; are easily torn, and the epithelial tissues more opaque than natural, and filled with granules mixed with oil-globules. The kidneys are somewhat similar to those in cases of scarlatina. In other organs the appearances are like those of ordinary acute uræmia. Secondary inflammations having sometimes existed, we may find evidence of them in the tissues affected. Urea is found in the blood in considerable quantities. Dr. Garrod found as much as 1·14 part per 1,000. It is readily found in blood collected from the right side of the heart.

The other changes possess no special characters. There may be signs of low inflammation of lungs, pleura, pericardium, and peritoneum.

and of much congestion of the lungs. The brain and membranes sometimes full of blood, as in low fevers, in which the blood is in fault. The blood coagulates loosely. Of course to these must be added gangrenous patches, bedsores, ulcers, ulcerated corneæ, boils, suppurated or swollen parotid glands, gangrene of lungs, ecchymoses, and purpuric or scorbutic patches, which occur occasionally.

PATHOLOGY DURING LIFE.—From the study of Cholera as shown in the symptoms and post-mortem appearances, we pass to an attempt to account for them. In the section on Etiology the nature of the cause has been discussed. We have now to suppose that a poison has entered the blood in some manner, either by the channel of the lungs or intestinal surface. The poison acts through the blood, possibly in the manner of a ferment; but we do not know whether it acts by a catalytic influence on the blood mass, or whether it multiplies itself in the blood, or not; we are very much in the dark. However, if we cannot trace the minute action of the poison we are able to guess at some of its effects. It appears that at least two great sets of capillaries and small arteries are involved in them: those of the lungs and intestines. They seem to be very differently influenced by it. In the lungs very little of the blood passes freely through them in the algide stage. In the intestines an enormous quantity of certain of the blood elements passes through the capillary walls in the exudation period. In both of these sets of actions, parts of the nervous system appear to be under a morbid influence. In the lungs the muscular fibres of the small arteries seem thrown into a state of contraction. In the intestines a sort of paralysis of the smaller arteries and capillaries seems to exist, much as occurs in the sections of the sympathetic nerve in the neck in Bernard's experiments. That the morbid action in the lungs is of nervous character seems probable from the absence after death of any discoverable mechanical obstacle to the passage of the blood, from the paroxysmal nature of the dyspnœa at first, and from the ease with which the pulmonary circulation is re-established when recovery begins. The nervous character of the actions in the intestines seems probable from its analogy with the results of Bernard's experiments on the sympathetic, in some of which a section of that nerve in the neck caused the surface of the skin supplied by the vessels under its influence to become bathed in sweat. It is a matter of dispute whether the disturbances in the lungs and the exudation through the intestinal walls are both effects of the poison, or whether, exudation into the intestinal canal having taken place, the pulmonary symptoms may not be due to the altered condition of the blood, caused by its being inspissated through the exudation of so much of its fluid and saline elements. It must be granted that symptoms similar to collapse may be produced by poisons without any purging. I have seen people under the influence of malarious poison in Calcutta lie for hours as cold, and pulseless, and as embarrassed in the breathing, as in Cholera. In gangrene, too, a somewhat similar condition may come on. It appears, then, that symptoms similar to those of the cold stage of Cholera may be produced in certain diseases without any exudation or loss of fluid from the blood. Nevertheless, it is doubtful whether we can say positively that exudation is not necessary to produce the collapse in Cholera. It is true that it is said that some of the most rapidly fatal cases of Cholera are those in which there is no purging, and that therefore we must seek for some other cause of the algide stage. But it does not follow, that because there was no purging there was no exudation into the intestines. The exudation is sometimes poured out, and retained there, and we should never for a moment confound transudation with purging. In numerous instances in which there has been an absence of evacuations, post-mortem examination has shown the intestines to be full of fluid. In others, the symptoms of full collapse have come on before the vomiting and purging. Among these a remarkable case occurred in the Middlesex Hospital, and is reported by the Committee for Scientific Inquiries in the Cholera epidemic of 1845. A child was admitted in a state of collapse, having had only one motion and vomited a little. Soon after admission it was copiously purged. In this as in the other cases, the exudation had taken place, but had been retained, and but for the evacuation, after the algide stage had fully formed, might have been set down as a case of collapse without transudation. In many cases reported to have died or to have passed into collapse without evacuations, we find the symptoms before collapse to have been similar to those caused by some drain upon the system, as has been mentioned under the head of "Varieties" of the disease. It certainly is not common for the practitioner in India to meet with these cases of absence of evacuations. Every now and then one hears the patient's friends say that such an one threw up a little water from his mouth and sank into collapse, or died at once; but in actual practice they are not often met with face to face, as far as my experience goes. Further careful observations are required to be made upon these cases, in which after death the contents of the intestines should be measured. It may after all turn out that the supposed absence of discharges may be in cases in which there is retention and not absence of exudation. The rapid improvement that follows injections into the veins in collapse strongly supports the view of the dependence of this upon the loss of fluid from the blood. It is hardly possible that this resuscitation of the patient from the depths of exhaustion can be accounted for, as Dr. G. Johnson contends, by the warmth of the injected fluid. Although the subject still demands careful observation, there seems great probability that the collapse has a relation to the transudation, if not to the purging. The cases which at present most oppose the view are those in which the body remains rather plump in the collapse stage; but I think that in these there is generally little sweating, and therefore there exists one drain the less for the superficial capillaries. I am far from denying the adequacy of the Cholera poison to produce the algide stage by its action on the vessels of the lungs, or by a sedative influence upon the centres of circulation and respiration, though I think there is not yet full or complete evidence of it.

It has been argued that the vomiting and purging are salutary, and that they eliminate the poison. It is very questionable whether fluxes produced by organic poisons are necessarily eliminative of the actual poison that was introduced into the system, or of its products. Whether the exudations in Cholera are eliminative or not, there can be but little doubt that they are very destructive methods of cure. If this purging were beneficial, we should expect to see that cases of Cholera with preliminary diarrhœa would be slight; whereas we often see that a man has diarrhœa for a week or more, quite as copious as could be produced by a few doses of castor oil, and yet these very cases often pass into profuse purging, collapse, and death. Most men who have had much experience in Cholera will say that when they succeed in checking the discharge, before collapse comes on, their patients are saved. They justly fear the result of active purgative medicines given in seasons of Cholera. It is hardly possible that experience can have gone quite wrong in these matters. We do not know how the poison may be eliminated. For aught we know, it may be decomposed in some way, and not eliminated at all in its entire state.

In collapse the patient's state is a remarkable one. Circulation is nearly stopped, and indeed all vital functions reduced to a minimum, as shown in the description of the symptoms. Little or no natural chemical action goes on in the system; but it does not follow that some chemical changes do not go on in the blood itself, as they might do out of the vessels at a similar temperature, and in prolonged collapse this may furnish some impurities to the many mingling with it. The altered density of the blood consequent upon the loss of so much water must have brought about many changes, involving both the intercellular fluid and the blood-globules, and materially affecting the relations between them. Schmidt supposes that after about thirty-six hours the morphological elements of the blood are nearly one-half more than the normal proportion. The proportion of inorganic salts is not increased, except perhaps for the first four hours, during which the water passes out more rapidly than the salts; after eighteen hours they sink much below the natural standard, and still more in thirty-six or forty-eight hours. The result is that the proportion of albuminates in the blood-globules is increased by one-half, and in the intercellular fluid to double of what would be normal. He considers that the solids are retained in the vessels with more force than the water, the organic solids with more force than the inorganic, the phosphates with more than the chlorides, and the salts of potash with more force than the salts of soda. In the exosmosis from the globules into the intercellular fluid, he supposes that the same laws of the succession of diffusion of their contents prevail; the chlorides of the alkalies being the first salts to transude both from the intestinal capillaries and from the blood cells into the liquor sanguinis. It is probable that the blood, thus altered in the relations of its component parts one to another, does not recover its natural state at once, even after fluid has been absorbed into it during the reaction stage, and that the abnormal state thus produced may help to cause some of the consecutive disturbances.

In healthy reaction, full purification of the blood by the liver, kidneys, and intestinal surface occurs, and this is promoted by free absorption of water; and the normal composition of the blood is further brought about by absorption of the saline matter still lying in the intestines or administered purposely. Dr. Morehead has observed that the more rapid the reaction without return of the secretions, the greater the probability of secondary fevers, &c. Doubtless, the more rapid the reaction the more rapid the tissue changes, and the more rapid the accumulation of effete matter in the blood; and hence the greater urgency that there should be free secretion and excretion. Some writers attribute the consecutive morbid actions, or at any rate a great part of them, to the stimulants wrongly given in the collapse stage. Injudicious stimulation and drugging may certainly still further poison the blood by themselves, or, by inducing over speedy reaction, they may assist in bringing on fevers; but I have seen a very large number of people suffer from secondary evils, who have undergone no treatment whatever, and am convinced that the natural progress of the disease is, of itself, sufficient to bring them on. Judged by the colouring matter in the intestines, the functions of the liver appear to be more readily restored than those of the kidneys. Disturbances of the liver are rarer than those of the kidneys. We can scarcely expect the secreting functions of any of the organs to be restored immediately under any circumstances, and some hours must be allowed for the return of normal action. Prolonged cessation of urinary secretion, of course, ends in coma and death. In some of these cases the worst symptoms may be delayed by the urea being excreted through the surface of the intestines and stomach. It is probable that in some of the consecutive fevers, without there being a suspension of the separation of the urea, there may be some diminution of the amount passed in the twenty-four hours, and there may be at the same time a loading of the blood with creatine, creatinine, and other effete matters. To some of the forms of blood-poisoning are due the parotid swellings, purpuric spots, boils, and aplastic inflammations of various parts. To the deficiency of available elements in the blood is due the defective nutrition of the cornea, and perhaps the gangrenes. I have not seen any reason to suppose that any of the last are caused by embolism. The causes of the dyspnœa may be two—spasm, or constriction of the smaller branches of the pulmonary artery and collapse of the lung tissue. It is probable that constriction of the arteries may frequently be the cause, especially in the instances in which the embarrassment is temporary and paroxysmal, one set of arteries being contracted at one time, and one at another, in areas of varying sizes. It seems likely that the permanent dyspnœa may be caused by collapse of the lung tissue, especially in the later stages of the disease.

Collapse of lung tissue is not necessarily permanent, but the solidified part would probably continue unexpanded in the feeble inspiration of the stage of exhaustion, if any considerable area of the lung were affected.

The purpuric conditions, besides the causes already mentioned, may be produced by large doses of calomel. When this was used in scruple doses to check vomiting, it was not uncommon to give two or three such doses. One can imagine that forty or fifty, or even fewer, grains of calomel, accumulated in the stomach or intestines, might be absorbed in reaction, and be ready to add its liquefactive action to the other blood-dissolving agents already at work. Cases of this sort, which I have seen, have made me cautious about the use of large doses of mercury in Cholera.

The anæmia and debility which succeed Cholera are readily accounted for by the profound disturbance to which all the blood elements have been subjected, and which may destroy blood-globules, or interfere with their renewal.

Chronic diarrhœas and dysenteries may depend upon the irritation which has been set up in the small and large intestines respectively; producing in some instances ulceration and softening of the mucous membrane. The softening of ecchymosed blood may also produce symptoms of diarrhœa or dysentery, by giving rise to patches of inflammation or disintegration of the mucous membrane.

I once saw a case of chronic albuminuria which appeared to have originated in an attack of Cholera a year previously.

PROGNOSIS.—*Favourable signs.*—In the *Diarrhœal Stage:* The not passing rapidly into collapse, the pulse keeping some strength, the countenance being tranquil and natural. In some cases the prolonged diarrhœal stage may be fatal without collapse. These are probably choleraic diarrhœa.—In the *Collapse Stage:* The collapse not becoming deep, the pulse remaining in the brachial, respiration tranquil or not much embarrassed, without deep lividity, the algide period not lasting beyond six or eight hours, the cessation of jactitations, and the tendency to quiet dozing or to snatches of easy sleep, gradual return of circulation, progressive improvement in strength even if gradual, milkiness or greyness of stools. The continuance of scanty stools is not of much importance; if the belly is at all tumid, they are advantageous, because they free the intestines of matter already exuded into them. The more mucous and gelatinous they are, the better it is that they should pass away. Cessation of vomiting and purging without reaction after a little time is not important.—In the *Reaction Stage:* Early establishment of the secretions, within twenty-four or thirty-six hours, refreshing sleep, and tolerance of food and drink.

Unfavourable signs.—Quick disappearance of pulse from the radial or brachial arteries, early embarrassment of respiration, and lividity of surface, profuse sweating. In the *Reaction Stage:* Suppression of urine, advent of typhoid symptoms, bloodshot eyes, secondary inflammations.

Coma is always dangerous. Want of progress in the symptoms of improvement, and continuance of diarrhœa or vomiting, are unfavourable. Pink or bloody stools or hæmorrhage from the stomach are almost always fatal signs. Disproportionate muscular strength without corresponding improvement in other functions is not necessarily favourable. Allusion has already been made to this under the head of "Varieties."

TREATMENT.—This is most successful when commenced early; before collapse. In full collapse it is pretty certain, as remedies cannot be absorbed, that they cannot be of much use. Still, it must not be supposed that all treatment is useless in Cholera, or even that it is useless in all the states of the stage of collapse. It cannot do much good in perfect and complete collapse, but in all the other stages of the disease it is as beneficial as in any other severe and dangerous illness. Excluding collapse, judicious management, free from routine, and adapting the treatment to the individual case, is of great importance. Cholera is not merely a disease of vomiting and purging; and this will have been apparent from the foregoing pages.

The treatment should be considered in relation to the evacuation, collapse, and reaction stages.

Evacuation Stage.—The discharges should be checked, if possible. I believe that the great object of treatment is to restrain the passage of exudation from the blood into the intestines. The remedies used for this generally check the vomiting and purging, so that in the condition of these, taken with the state of the pulse, we have a sort of indication of the degree in which the transudation is interfered with; a sort of indication, because there is not always any close relation between the time of the transudation and the discharges from the bowels. The first medicine given should be a full dose of opium—to an adult 2 grains; in India, 1 to 5 grains of calomel are generally combined with it. I do not know that the calomel does good; it does no harm. If this dose is retained, probably little more medicine will be required. We must not expect the purging to cease immediately that the medicine is swallowed. Perhaps one or two motions will pass away after the dose has been given. Soon after the opiate, if it has been retained, say in half an hour, in my own practice I give an astringent, generally the following mixture:—℞ Plumbi acetatis, gr. xxx; acid. acet. ♏x; aquæ distillatæ, ℥vj—1 oz. or ½ oz. every half-hour or hour. At the end of an hour from the administration of the first dose of opium, if the purging persists, I give one grain of opium and continue the astringent. The sugar of lead mixture may be given every half-hour or hour, if the purging continues smartly; but if it seems inclined to cease, and the pulse keeps good, the mixture may be left off, or given at wider intervals. It is necessary that the medicines should be presented to the stomach in the simplest and least irritating form. In the form of pill, opium is probably more easily retained than laudanum, which often nauseates; but when a pill is not at hand, forty minims of tincture of opium should be given in a little cold or iced water, or in a little brandy. The acetate of lead mixture is nearly tasteless, or may be made so by the addition of a little iced water. Dr. Graves's acetate of lead pills may be given instead of the mixture, but are probably less active than the liquid medicine. These pills are made as follow :—Acetate of lead, 20 grains; opium, 1 grain, divided into twelve pills, of which one may be given every hour. It would be better to increase the lead to 2 or 2½ grains in each pill. Tannin or gallic acid may be given instead of the lead, but they are more likely to cause sickness. If the above medicines check the discharges, all danger will probably be over in a few hours. Consecutive disease is not likely to follow such a simple case. When there is vomiting there is more difficulty. The pill should be given as before. If rejected in the course of a few minutes, we shall probably find it in the discharges. It will be well to wait ten to fifteen minutes before giving more opium, and (while waiting) to apply a large sinapism to the epigastrium. If we are satisfied that the first pill has been rejected, we should repeat the dose of two grains in about a quarter of an hour after the vomiting. If we are uncertain of this, we should in half an hour give one grain only. After removal of the sinapism, if the stomach is quiet, the acetate of lead should be given in mixture or in pills. Perhaps half-doses of the mixture may be retained if the full quantity is not. When the stomach is very irritable, a solution of half a grain of muriate of morphia may be injected hypodermically. When simple opium cannot be tolerated by the stomach, hydrocyanic acid and other anti-emetic drugs are rarely more efficacious. Stimulants are not wanted in this stage unless the pulse begins to flag ; then a little brandy and water, or, better still, iced water, should be given, a teaspoonful or two at a time. Champagne is sometimes tolerated, when other wines are not retained. In the majority of cases a little brandy diluted with ice dissolved in it, or with plain cold water, is the best stimulant. Cramps are generally relieved by friction with chloroform, or with the hand ; if severe, by the inhalation of small quantities of chloroform. The doses of opium recommended may of course be modified. If the symptoms are mild, one grain may be given instead of two. Some prefer to give smaller doses every half-hour or hour, but it seems better to give a decided dose at the onset, and not repeat it often, and not to give more than three grains in all, within the first three hours, unless the medicine has been vomited; then the doses should be managed so as to make good the quantity supposed to have been rejected. As a general rule we should limit the quantity of opium to three grains and the acetate of lead to ten or fifteen grains in the first three hours. If the disease should show signs of yielding, we can diminish the doses, or stop them altogether. In some cases, if collapse does not come fast, the evacuations still proceeding, the opium may be continued in half-grain or grain doses about once in three or four hours for two doses, beginning three hours after the third grain has been given. The lead mixture may be continued in half-ounce doses every two hours for five or six doses if necessary. When the collapse is progressive, no opium should be given after the third grain, and, indeed, if it seems to be approaching fast even the third grain should be withheld.

Collapse.—When this is complete, opium should be entirely avoided, and sugar of lead would be useless. The exudation is probably quite stopped, and the evacuations now passed are merely residues of matter previously poured into the canal, and add nothing to the danger or exhaustion of the patient. In the worst stages of collapse, stimulants are of little avail. Æther and ammonia properly diluted may do no harm. If they can be retained, small doses may be given every half-hour. A little weak brandy-and-water is sometimes retained better than any other stimulant. But the administration of stimulants in the stage of collapse requires great care and discrimination. If they are of use, they make themselves felt in the pulse. If it revives under them, ever so little, and falls back or disappears in their absence, they may be continued cautiously; otherwise they, or at all events the alcoholic stimuli, should not be persevered with. The dependence of the pulse upon the stimulants is certainly sometimes seen. Unfortunately, if stimulants, such as brandy and wine, do not act beneficially, they are likely to be hurtful in the after stages. A large quantity of them may be collected in the stomach or intestines, and when absorption begins they may be taken up into the circulation, causing undue rapidity of reaction before secretions can be formed, and so adding to the mischief already at work in the blood. So that we may be pretty sure that if they do not work for good they will be hurtful. Nothing is more pernicious than the system of pouring large quantities of brandy into a pulseless patient in Cholera. Sometimes when the irritability of the stomach has gone off he will, in the course of two or three hours, swallow a large quantity of brandy, water, æther, ammonia, sago, wine, &c. and the friends perhaps congratulate themselves upon the quietness of the stomach; the pulse remaining absent nevertheless. In the course of time the patient grows very uneasy, and presently vomits the whole accumulation, perhaps two or three pints. Such cases as these show the uselessness of overloading the stomach in the torpid condition in which the patient is in collapse, and what a reservoir of mischievous elements may be provided against the return of the circulation. We often see cases in which stimulants, mixtures, &c. are given and as constantly vomited. These are often instances of too much medication. The irritable stomach is made more so by the drugs, and on leaving these off the vomiting ceases. The dependence of the vomiting upon the time of giving the doses should always be inquired into. We ought not to take it for granted that the vomiting is always the effect of the disease; it may, and I have so seen it scores of times, be due to the administration of remedies. A few teaspoonfuls of brandy-and-water and a few doses of æther and ammonia will do no harm in the pulseless state, but it is vain to expect to do good by constantly pouring them down the patient's throat. The best thing for him in the stage of full collapse is to gratify pretty freely his instinctive desire for cold water. This cannot do harm: a little may run off by the bowels, or be vomited, but if any remains to be absorbed in the reaction stages it will have the best possible effect. It is of no use to keep up the patient's vomiting by giving him as much as his morbid thirst impels him to swallow; but a considerable quantity may be given in the course of two or three hours by tablespoonfuls or wineglassfuls at a time; the colder the better. If ice be available, it will be better to give iced water, or to place pieces of clear ice in the patient's mouth frequently. This quenches his thirst, and seldom causes vomiting. There need be no restriction placed upon the quantity of water given beyond that it is advisable not to provoke too much vomiting by it. Although I have urged the uselessness, nay mischief, of pouring down drugs and stimulants into a patient in the state of perfect collapse, there are certain conditions of collapse in which when given cautiously they may be of great use. The cases are those in which collapse is not perfect, in which the circulation still goes on, though feebly, throughout the algide stage. In these the volatile stimuli, æther and ammonia, in mixtures, and weak brandy-and-water, are certainly beneficial. They maintain the strength, as they do in any other diseases with exhaustion. Ten to twenty drops of liquor ammonia, or thirty of sulphuric æther properly diluted with iced water or cold water, given every half-hour or hour, with an occasional tablespoonful of brandy-and-water, may be continued while the pulse seems to be maintained by them. In these cases the Cholera mixtures and Cholera pills so often used in India are of service. They are made up in various ways, but contain generally such stimulants as æther, ammonia, tincture of camphor, and essential oils, as cajeput, aniseed, cinnamon, peppermint, &c. or some of them. Most of them also contain laudanum, but it would be better to omit this, and add it to the dose when considered necessary. Cholera pills can be made up of camphor, two grains; asafœtida and long pepper, each one grain, with a little mucilage. A pill should be given every one and a half or two hours. They are useful in the same circumstances that the Cholera mixtures are. When acetate of lead has been used as the astringent, the liquor ammonia should be used in preference to the carbonates of ammonia, or their preparations. It will be observed that the principles advocated in the use of stimulants are that we should be guided in the administration by the pulse. While that remains, absorption and other vital actions go on, although perhaps in a diminished degree; and while these continue, stimulants may assist the patient through the crisis, and may prevent his passing into the pulseless state. The volatile and diffusible stimuli are less pernicious than alcohol, but a small quantity of the latter is useful, and should be occasionally given. There are few persons to whom three ounces of brandy may not be safely given during eight or ten hours if their circulation responds to it,—in some cases even more, but probably not often. As soon as it is found that the pulse maintains itself without them the stimulants should be withdrawn; the alcoholic first. Administered in this way they will not do harm; they are taken into the system by degrees, and their action is spread over a sufficient space of time, and they thus act very differently than they might do if allowed to accumulate in large quantities in the stomach, to be absorbed all at once and added in mass to the blood when the circulation is renewed. Many of the extreme collapse cases will come round by themselves as well as or better without stimulants than with them, and, therefore, we must not suppose that we are accelerating death by leaving the patient to cold water in such cases. For the sake of trying whether the pulse can be roused we may give a little stimulus now and then, taking care that the total quantities given shall be too small to do harm. I have made these observations because I think that there is a tendency to consider stimulants as inadmissible in the treatment of Cholera. Much mischief may have been done by them sometimes, but with due attention to the principles that I have mentioned for both opium and stimulants I think that good, not evil, may result. The discriminating use is very different from the abuse of remedies.

The profuse perspiration should be rubbed off the surface with a dry soft towel. In the Indian hospitals dry ginger powder is often used with friction for absorbing the moisture. If used, care should be taken that the powder is impalpable, and free from grit or dirt; otherwise troublesome excoriations will ensue.

Cramps are, as in the earlier periods, soothed by friction with turpentine and chloroform. Even in great debility, moderate chloroform inhalations may be used without increasing the weakness of the pulse. Food will not be borne, except, perhaps, in the prolonged cases of semi-collapse, but when signs of reaction appear, a little beef-tea or chicken-broth may be given. Starchy matters are apt to generate gas, but sometimes a little sago or gruel may be given with advantage, as they lubricate the coats of the stomach. When the pulse is very weak, in cases in which stimulants are given, a little port wine may be added to the sago or arrowroot.

Warm baths, or vapour baths, or medicated wet sheets, are of no use, and the patient is so restless, and throws himself about so much, that it is difficult to apply them.

Reaction Stages.—If the patient is regaining strength, and his secretions return, there is little to be done. He is best left to nature, a little liquid food, and cold water. To compensate for the loss of saline matter from the blood, a little common salt should be added to his food, and he may occasionally take a draught of water to a pint of which a drachm of carbonate of soda is added. This last also is grateful to the patient when he has heartburn or heat of stomach. It is not necessary or desirable to give a dose of aperient medicine within the first few hours of reaction in order to promote the secretions. The officious use of purgatives and mercurials, too soon, often brings on a relapse. If the bowels are confined, and there is tension and uneasiness, a warm water enema—say a quart of water with a little assafœtida, or sal volatile, or æther—will be the safest remedy.

Vomiting is often continued in the reaction stage, and may have been caused by the irritation left by frequent previous retching, or by excess of stimulating remedies. In these cases a single dose of opium or morphia—say a grain of opium—will often act admirably. It may be repeated every four or six hours if the vomiting continues. The food requires careful management. Sometimes a table-spoonful of milk with one of lime-water may be given every half-hour or hour for the first day or two, or a small quantity of ice-water only. It is desirable to give the stomach as much rest as possible. In some cases the irritation is due to subacute gastritis, and requires a few leeches to the epigastrium—a blister is useful in all forms of the vomiting in the earlier stages of reaction, and sometimes one-half or two-thirds of a grain of morphia may be applied to the raw surface with excellent effect. Food should not be pressed upon the patient. For days he may not be able to take more than tea-spoonfuls or desert-spoonfuls of milk and lime-water, arrow-root, barley-water, &c. In some instances it will be necessary to trust entirely to nutritive enemata. Effervescing draughts with excess of soda are sometimes useful, and hydrocyanic acid may be tried.

Diarrhœa, if slight, should not be interfered with at first, especially if the evacuations are at all offensive or bilious. Where decided enough to be weakening, gallic acid, or tannin, or mild opiates, or chalk mixture with carbonate of soda, should be given. If there be tenesmus, a warm water enema with a drachm of carbonate of soda should be used, and followed after operation by one of laudanum and a little water. Turpentine stupes should be applied to the abdomen in all the states of intestinal irritation.

Hiccup is frequently accompanied with much eructation of gas, and is difficult to stop; it is not dangerous. It may be treated by sinapisms to the spine, in the cervical and dorsal regions, and by ten minims of chloroform in mucilage every half-hour, or by chloroform inhalation, by morphia, or small doses of belladonna, and, if there be acidity, by alkalies. The common anti-spasmodics seem to have little effect.

Want of sleep, without pyrexia or heat of head, may be relieved by a dose of calomel and opium, and perfect quiet, hot pediluvia, and, if very obstinate, chloroform inhalation. Sometimes a mild dose of alterative aperient medicine is beneficial.

Imperfect reaction is very troublesome. Unless the discharges are arrested, the patient will sink in three or four days. A small dose of calomel and opium may be given. The following astringent mixture sometimes arrests the vomiting and purging: Gallic acid, 10 grains; tincture of opium, 10 minims; dilute sulphuric acid, 10 minims; water, 1 ounce. This should be mixed with a little cold or iced water, and given every four hours. Turpentine fomentations to the abdomen, or sinapisms; non-irritating food should be prescribed.

Champagne is sometimes useful. It is better not to give too much liquid, but thirst may be moderately gratified.

Uræmic Stage.—Purging should not be checked in this. It is exceedingly difficult to restore the renal secretion. The kidneys are gorged with blood abounding in urea, their natural stimulus, and yet they will not act. When it is seen that suppression exists, the patient should be encouraged to drink as much water as he can. It is the best diuretic; and sinapisms, dry cupping, or cupping, should be applied to the loins. He should take a drachm of liquor ammoniæ acetatis, half a drachm of spiritus ætheris nitrici, and 5 to 10 minims of tincture of digitalis in water, every three hours. The special symptoms of uræmia should be treated as uræmia generally, and need not be detailed here. When coma comes on or is approaching, it would be advisable, if the pulse is firm and good, to try the effect of a moderate venesection. This might relieve the renal congestion. There is no more unfavourable state to treat than that of approaching head symptoms, with vomiting of grass-green matter, and diarrhœa. The discharges are exhausting, and if stopped, coma is hurried on all the faster, because the urea is to a certain extent relieved by the evacuations.

Fevers.—The mild cases require little interference. They need only a small quantity of nourishing food, not forced upon the patient, perhaps a little alterative and mild aperient medicine, effervescing drinks with excess of alkali, and quiet. The more severe fevers need all the attention and prolonged care of severe adynamic fevers, and, according to the state, stimulants, wine, tonics, and nourishment; but there is nothing special in the treatment. The same may be said of boils, gangrenes, inflamed parotids, sloughing corneæ, purpura, &c.

Much pallor and debility is often left after protracted illness in Cholera. These demand quinine, iron, strychnine, and other tonics. Change of air is often the most efficacious remedy, and such a total change as a voyage from India to Europe, and residence there, is often demanded.

Throughout the collapse and subsequent changes of debility, the patient should be kept in the recumbent posture, and during the uræmia and fevers the most scrupulous cleanliness of the bed should be preserved, and no wet or soiled sheets allowed to remain unchanged. The sacrum and hips rapidly excoriate, and very troublesome bed sores are quickly formed. The surgeon should examine these points himself frequently. The hypogastric region should be examined twice daily in order that retention should not be mistaken for suppression of urine, a mistake which may now and then happen in the early reaction stages, and sometimes in the typhoid and comatose periods.

From the first to the last there is no disease which requires more careful attention during treatment than Cholera, or more watchfulness on the part of the surgeon. In the early stages it will be impossible to leave patients with mere routine directions for treatment if we wish to be successful. The when to do, and the when not to do, in a disease with such rapid changes as Cholera, requires much more discrimination and knowledge than is usually possessed by nurses or friends. The case requires throughout, the constant guidance and vigilance of the surgeon, and he must specially assume this action during the critical periods of the disease. Good nursing is always of the utmost value, but patients in Cholera need that this should be frequently superintended. In military and civil hospitals, especially in India, in which the nursing must be confided to native servants or to the comrades of the sick man, the surgeon is obliged to be doubly watchful.

All patients with Cholera should be treated in well-ventilated rooms or wards. On account of the sad spectacle presented to the patients with other diseases in hospitals, when a large number of patients are brought in for treatment, and to avoid all possible chance of diffusion of the disease, small though that chance may be, it is desirable that all Cholera cases should be treated in separate wards, and with ample space and ventilation about them. Should there not be such wards or rooms, tents should be used. I prefer rooms to tents if they are well-ventilated, and have non-absorbing floors and walls that can be washed with water and chemical solutions. Tents, unless they are thoroughly cleansed and purified, are as likely to spread disease as hospitals are. They are colder than rooms, and the attendants and patients themselves are likely to keep them shut up, and from their size the air within them is sooner rendered impure than in an airy ward. Those surgeons who remember the severity of hospital gangrene at Ferozepore, in the Sutlej campaign of 1845-46, know that tents present no immunity from hospital evils. The only real advantage of a tent is the power of changing its floor by shifting the ground, but against this are to be placed the coldness at night, and the discomfort and consequent diminished efficiency of attendants. Of course there are times when the tent must be accepted, but I should in preference select to treat my Cholera patients in airy wards, in which I could regulate the temperature, in which I could wash and scrub the floors and walls, which permit of economical use of nurses and attendants, and in which they have that comfort and warmth which promotes their efficiency. In speaking of tents, of course I refer to their use in warm climates; in cold climates, except in summer, they could hardly be used. In India, in the warm seasons, too, we are subject to heavy storms, which render tents most uncomfortable, and I have seen several tents, with wounded men, blown down in one night. Those only who have spent weeks under canvas in the North-West Provinces in India know how exhausting is a sojourn in them in the day-time, and how much cooler a well-covered house is. In the rains, too, they are most uncomfortable; either chilly in the heavy showers, or close and stifling if shut up. I have made these observations because I believe that there is a disposition to adopt tents on all occasions for treatment of Cholera. I think that this is an error. There are times when tents must be resorted to, and when to use them at all times and all seasons is the least of two evils; but I should use them in times of necessity only.

The duty of the medical practitioner is not limited to the treatment of cases presented to him. It is most important that all cases of diarrhœa, choleraic diarrhœa, and incipient cholera should be treated early, and the physician should take the initiative in impressing this upon all within his reach during cholera epidemics. Patients should be provided with instructions on the symptoms, and, when living in distant places, in addition, with remedies that they can use until efficient assistance can be had. Surgeons of regiments should organize daily inspections of the men in barracks, and all cases of relaxed bowels should be treated on the spot, and the patients not sent to hospital unless for real Cholera. The fear of being sent to hospital constantly prevents men from reporting these ailments, which they would readily do if it were not for the dread of being made in-patients. These cases can be readily treated out of hospital, and with the advantage of lessening the crowding of the wards.

TREATMENT OF CHOLERAIC DIARRHŒA AND DIARRHŒA.—The discharges should be restrained. A grain of calomel with a grain of opium morning and evening, and with a dose or two of chalk mixture and soda, or of tannin or gallic acid, with sulphuric acid in the middle of the day, will perhaps suffice, if continued for two or three days. With these should be combined turpentine fomentations to the abdomen, light digestible food, and if there be exhaustion wine with arrowroot or sago, or warm brandy and water occasionally. The patient should give up work, and keep to his bed in cold climates, or to his couch or sofa in warm ones. If there be rice-water stools the patient should be treated as for the first stage of Cholera. If the diarrhœa continues for several days in spite of treatment, the patient should, if possible, be moved altogether out of the locality in which Cholera prevails. Choleraic diarrhœa, like Cholera, is much more intractable and dangerous in the commencement of epidemics; following indeed the rule of Cholera itself. In these periods many cases run into Cholera in spite of early and assiduous treatment. The disturbances which sometimes precede both choleraic diarrhœa and cholera may be treated with mixtures containing sal volatile, æther, and aromatics, tonics, and regulation of diet. Many of these sensations will be due to the imagination, to fear, &c.

Cholera in children must be treated upon the same principles as in the adult. Laudanum must be given in preference to solid opium. It is necessary to be very cautious about the repetition of doses of the opiates. Indeed, unless the first dose be vomited directly, it had better not be repeated for the young child under three quarters of an hour, or an hour, and not at all if there be any signs of drowsiness. The best stimulants are sal volatile and tincture of camphor given in a little iced water or thin arrowroot; a few drops of port wine or brandy in arrowroot may be given occasionally. In reaction it will be better not to give it the breast for twenty-four hours, but to feed it instead upon milk diluted with water and a little lime-water. The mother's milk if given should be pressed from the breast and diluted with a little water; or, if available, a little donkey's milk may be given. If the child has been in the habit of taking broths or farinaceous foods they can be given in small quantities, and not strong.

It would be vain to attempt to give an account of all the plans of treatment and remedies proposed for Cholera. They are of opposite character and very numerous; two or three deserve notice.

Venesection.—This was formerly a good deal used in India, but is not often resorted to now. In the spasmodic febrile forms Mr. Twining bled, and he thought with advantage, in the early stages. In collapse little blood flows, and on the whole probably when it does the abstraction does more harm than good.

Saline Injections into the Veins.—This, when first introduced, seemed of brilliant promise. From the extreme of collapse the pulseless man rapidly recovered strength and sat up in bed, and talked, and looked himself, but the amendment did not often last; purging returned, and with it the collapse. No means have been found of keeping the injected fluid within the vessels; alcohol, quinine, laudanum, albumen, and other things, have been tried but have failed, and from this the injection plan has just missed being a great and glorious discovery. The following formulæ for the solution have been recommended:—By Dr. Latta, carbonate of soda, ʒj.; muriate of soda, ʒiij.; water, 6 pints. By Schmidt, chloride of sodium, 60 parts; chloride of potassium, 6 parts; phosphate of soda, 3 parts; carbonate of soda, 20 parts—all by weight: 140 grains of this mixture to be dissolved in 40oz. of distilled water and filtered. The temperature of the injection should be about 108° to 110°, and the specific gravity 1005 or 1004. An endeavour is made to make a solution of similar composition to the fluid lost by the blood. It should be injected slowly, not above 40 or 60 ounces at a time, and not faster than at the rate of 2 oz. per minute. If purging and collapse return the injection can be repeated. In some of the cases it was repeated several times. Dr. Owen Rees thought that a fluid of higher specific gravity than that usually injected should be tried, and recommends the following:—Chloride of sodium, ℥iij.; phosphate of soda, ℥j.; carbonate of soda, ℥jss; sulphate of soda, ℥ss. To be dissolved in distilled water at 98° F. making the fluid of specific gravity of 1030. This solution would seem to be too dense.

Most of the cases on which the experiments were tried were in a state of extreme collapse, but the recoveries, as shown by Dr. Wright, Dr. Mackintosh, Mr. Twining, and others, do not show that more escaped than might have been expected if they had been left to themselves.

Inhalation of Oxygen.—The patient cannot use any apparatus so as to inhale during collapse; therefore any gas used must be brought into contact with the air cells by some form of artificial respiration. It is doubtful, however, whether oxygen would act upon the blood in its altered state. Some experiments have been made of impregnating the atmosphere with oxygen gas by decomposing chlorate of potash at the bedside, but no real benefit seems to have resulted.

Saline Plan.—Dr. Stevens recommended draughts of fluids containing chloride of sodium, carbonate of soda and chloride of potassium in order to supply the loss in the blood; but it has been of no avail, the mixture would not be absorbed, or, if absorbed, not retained in the blood.

Dr. Ayre's Method.—This consisted of administering 1 or 2 grains of calomel and 1 or 2 drops of laudanum every five or ten minutes for several hours, the object being to bring on a secretion of bile. This plan has not proved successful in the main, though, at the time of its introduction in England, some practitioners thought it beneficial.

PROPHYLAXIS.—Whatever may be our views as to the contagion of Cholera, it is well to act upon the principle that it may under certain circumstances be spread in a locality, and thus be upon the safe side. We ought not to neglect the evidence brought forward by Dr. W. Budd, and at all events act as if the fæcal discharges may become injurious to the healthy. They should never be thrown into the common privy, but be treated with chloride of zinc, and buried in trenches out of harm's way, at some distance from habitations, and where no drainage from them may find entrance into wells or cisterns. The bed-pans and other vessels used should also be washed with chloride of zinc, and thoroughly cleansed. The patient's bed should be covered with a waterproof sheeting to prevent the soaking of the mattresses, and the sheeting should be well cleaned with disinfecting materials. Better still, in hospitals the beds should be made up of soft straw, and when once used by a patient, the straw should be taken out and burned. The covering, the bed clothes, and all linen should be boiled in alkaline lye, and well washed. I believe that there is not the slightest necessity for burning the linen used. I believe that Cholera patients may be safely nursed by their relatives or friends, and that they run no unnatural risk in attending to them. Nursing mothers should not resume suckling their children, even if their milk returns, until several days after convalesence; the milk having been previously drawn off by some apparatus for the intervening days.

Removal out of an Infected Locality.—When practicable it would be well for people, whose occupations permit them, to move out of diseased places, but that can never be done on any considerable scale, except in the cases of troops. Under present rules issued by the Government of India, all troops are to move out of stations if epidemic Cholera prevails among them. This is therefore no longer a question for the consideration of the medical officer; he has only to do with the time of moving. Hitherto the experience of removal has been favourable as far as the diminution of cases and cessation of the disease are concerned; the deaths to cases have not diminished. Without doubt there are certain risks to be encountered in moving out, but they are smaller than the dangers left behind. The risks will vary with the weather and the seasons; but these moves have sometimes been made even in hot weather and rains, without increase of other sickness. These are the cases in which the discomforts and risks of tents must be accepted as preferable to remaining in the midst of the poison. It would be better to have lived under a tree or an umbrella than in some of the hospitals during the late epidemic at Meean Meer. It is not necessary to move far; a few miles are sufficient, and the march should be at right angles to the wind. Of course, in removal into camp, there should be realized absence of fatigue, good food, pure water, and thorough camp conservancy, or else there is no reason why troops should not suffer just as much from the disease as the Madras regiments seem to do, and as the regiments marching down from the hills to the siege of Delhi did in 1857.

Good food and pure water should be especially attended to in Cholera epidemics. Even without these being more than predisposing causes, there is sufficient necessity for attending to them. All indigestible food and all tainted articles should be specially avoided. It is well to bear in mind the teachings of Dr. Snow and Dr. W. Budd; and whether we believe that the exciting cause can be conveyed into the system by drinking water, or not, take care that all water consumed be entirely free from any fæcal pollution. On the same grounds that all food or water likely to produce irritation of the intestines, and thus determine choleraic action, are to be avoided, we should be extremely cautious about the administration of purgative medicines in the times of Cholera epidemics. Of course, occasionally they will be required, but they should not be given without decided necessity, and then never of the saline or hydrogogue character, and never at bedtime.

It has not been intended to give in this paper a full account of the hygienic measures required in the Cholera epidemics, and for lessening their severity. Fortunately these are fully treated of in Dr. Parkes's recent work on Practical Hygiene, Dr. N. Chevers's Review of the Means of Preserving the Health of European Troops in India, and the Report of the Commission appointed to Inquire into the Cholera Epidemic of 1861 in Northern India, which last may, I believe, be obtained in India. These from their nature are mostly applicable to troops, but their teaching may be made equally useful for all classes and for all communities.

CONSTITUTIONAL SYPHILIS.

By Jonathan Hutchinson, F.R.C.S.

The specific fever known as Syphilis differs chiefly from its congeners in the much more prolonged duration of its several stages. Like small-pox, measles, scarlet fever, and the others in this group, it is communicable from the diseased to the healthy, and can be produced by no other means; like them it has its several stages of incubation, efflorescence, relapses, decline, and sequelæ; as in them, so in Syphilis, the most prominent symptom is an exanthem, or cutaneous rash. The various stages of Syphilis tend to pass away of themselves in the course of time, just as certainly as do those of small-pox; and a well-developed attack affords for a time immunity from a second. As is the case in the other zymotic diseases, the poison of Syphilis is one which possesses the power of breeding in the patient's body, and the smallest possible quantity of virus suffices in due time to inoculate all the solids and fluids of the system. The time required, however, is much longer, and the stages are much more protracted. Instead of counting the duration of the stages by days, we have to count by weeks, or by months. From this circumstance there follow in the most natural manner certain apparent differences between Syphilis and the other fevers. Thus, because the evolution of the exanthem is slow and gradual, the pyrexial disturbance attending it rarely rises to any great height; because each stage is so much longer, correspondingly wider margins of occasional variation in length must be allowed. It further follows that because the disease extends over years, its subject is often not incapacitated by it for social life; many while still infected become parents, and transmit their own taints to their offspring, a circumstance which can but very rarely happen in the more short-lived and acute fevers.[1] These apparent differences are by no means real ones. Nor is it probably by any means correct to allege that Syphilis is the only fever which has a tertiary stage. What are called the tertiary symptoms of Syphilis find their analogies in many cases of small-pox or scarlet fever, in what are known as the sequelæ of those diseases. It is true that these occur only in a small proportion of cases, but the same holds good of the tertiary syphilitic phenomena. If we observed more carefully, it is probable that the sequelæ of the exanthems might be recognised much more often than they now are, and that many of the diseases classed as "strumous" inflammations of the eye, the ear, or the skin, with also diseases of bones and joints, are in reality the tertiary consequences of some specific fever. So, too, if it be alleged that the stages of Syphilis may be shortened and otherwise modified by treatment, while those of the other exanthems cannot, we may reply that those of the latter are too short and transitory to give time for a fair trial of remedies, and also that the power of remedies over syphilitic phenomena have probably been much exaggerated. Syphilis does fortunately differ from most of the other specific fevers, in that its virus is incapable of diffusion in the atmosphere, and that consequently it is contagious only, and not infectious also, but it is by no means solitary in this feature.

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[1] Hereditary transmission happens in all specific fevers if it chance that offspring are produced whilst the parent is suffering.

Having thus insisted upon the rank which true Syphilis ought to occupy in our nosological classifications,—a point of the utmost importance to our correct appreciation of its nature,—we may next ask the question, whether under this name we have to deal with one disease or with several. Every surgeon is aware that there are many venereal sores which are not really syphilitic, and very different opinions have been entertained as to the relation which these hold to the genuine disease. Some have assumed, with Carmichael, that there are several distinct poisons, and others, especially a modern and now very extensive school, hold that there are certainly two. Without entering at length into the controversy, I will simply remark that the evidence in favour either of plurality or duality,[1] is to my mind quite inconclusive. Let us accept clearly the doctrine so essential to the explanation of numerous pathological phenomena, that all living pus is contagious, and is capable of producing an inflammation similar to that in which it originated, and we shall not have much difficulty in explaining the different forms of venereal sores. The majority of the latter are probably *abortive inoculations.* In the performance of vaccination, the utmost care is taken to secure a pure virus, yet every now and then an abortive sore is produced. If it were the practice to inoculate again from these abortive sores when suppurating, we might soon produce an analogous state of things to what we now have in respect to the soft and hard chancre. The inoculation of Syphilis is of course a matter of mere chance, and the virus with which it is effected can be but very seldom in a state of purity. How rarely in the female do we meet with a healthy type of the indurated sore? Then, too, we must remember that this stage of the primary sore is transitory, and those who have once had it but seldom have it again. A large proportion of the women by whom contagion is communicated have had Syphilis long before, and are now no longer capable of originating the true virus. The sores which they possess are analogous to those caused by vaccination in protected persons. Attention to these considerations would, I feel confident, enable us to put aside the unnecessary hypothesis of duality.

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[1] The doctrine of so-called "duality of Syphilis" seems to me to rest on the most unsubstantial foundation. Surely it is absurd to speak of the "duality" of things which have scarcely any features in common. The production of constitutional phenomena is the essential feature of Syphilis, and there are not two forms of Syphilis as thus denoted. We may freely admit that, in impure sexual intercourse, pus of very various qualities and endowments may be inoculated, and thus very different kinds of local ulcers may result, but there is only one which can produce Syphilis.

In a medical essay on Syphilis any detailed consideration of the primary symptoms would be out of place, since these by common consent come under the care of the surgeon. We will content ourselves by saying that a successful syphilitic inoculation is denoted by the formation of a base of induration beneath the abrasion first noticed, that this induration usually occurs within a week of the contagion, but may be delayed for several weeks; that it is usually attended by very little either of ulceration or suppuration, and that it causes an enlargement of the proximal lymphatic glands. The bubo shows the same tendencies as does the chancre. There is hardness with but little swelling, and rarely much tendency to the formation of pus. These characters will be the same, or nearly the same, in both sexes, at all ages, and on all parts of the body.[1]

Stages of Syphilis.—A successful inoculation having been effected, a period of incubation now ensues, which may last from one to three months, and usually averages about six weeks. During the later part of this period, the patient is often sensible of slight malaise and discomfort. At length an exanthem makes its appearance, affecting both the skin and the mucous membranes. The skin shows a scattered eruption, which may vary very considerably in some of its characters, but usually conforms to rule, in being of a peculiar coppery hue, and in preferring the flexures of the limbs to their dorsal aspects. This rash may be merely congestive, resembling that of measles; it may be scaly, papular, eczematous, pustular, or bullous. In some few cases it is attended by ulceration, but as a rule it involves only the superficial layers of the skin, differing in this respect from the tertiary manifestations. There is not the slightest reason for believing that these differences in the character of the exanthem imply difference in the nature of the virus. Chancres exactly corresponding in their characters may be followed by most diverse kinds of eruption. Simultaneously with the eruption on the skin, we usually observe evidences of similar implication of the mucous surfaces. In the tonsils symmetrical ulcers form; these ulcers are of kidney shape, have a tawny grey base and abrupt edges. They are attended by but little pain, and do not spread either laterally or in depth. After lasting for a few weeks they usually heal. These tonsilar ulcers are rarely absent in the exanthematous stage. Very often, but not nearly so constantly, we observe also certain superficial patches of inflamed mucous membrane in the mouth, on the pharynx, palate, tongue, or cheeks. On the tongue, and at the corners of the mouth, and sometimes in other positions also, these patches become very considerably raised, and assume the condition known as condylomata. If condylomata be observed in the pharynx, they will almost always be observed at the anus also, and *vice versâ.* They are to be regarded as patches of cutaneous eruption modified by their position, and by the moisture of the parts. Whether or not in these cases any eruption extends through the alimentary canal, is a point upon which we have no evidence.

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[1] Some writers hold that certain parts never exhibit the phenomenon of induration when inoculated. My own experience is, however, very decidedly in favour of an opposite view. I have seen chancres with well characterised induration on the most various positions, *e.g.* the glans penis, the nipple, the lips, the eyelids, the hands, &c. &c.

The exanthem usually takes from a fortnight to a month before it is fully out, and about two months are usually occupied in its gradual decline. In some cases it is very transitory, and in some it is greatly prolonged. When it is at its height, or just when it begins to decline, it is not unfrequent for inflammation of the irides to occur. The iritis, when it happens, is usually symmetrical; it is attended by the free effusion of lymph, often in elevated nodules of a salmon or rust tint, the characteristic zone of ciliary congestion is usually well marked, and there is often a dotted deposit in the posterior lamina of the cornea. In other cases, instead of iritis, or coincident with it, inflammation of the retina occurs. The retinitis is, I think, usually a little later than the iritis, and we rarely see it until the eruption on the skin is well disappearing. This form of retinitis is not uncommon, and is a very insidious and most important malady. The patient notices nothing, excepting that his sight is very dim; he has no pain, no congestion of the front of the eye, no intolerance of light. The ophthalmoscope shows us the retina hazy, and as if stained with port-wine; the optic disc swollen and its margins indistinct; whilst not unfrequently numerous small extravasations of blood are seen.

Whilst the local phenomena just mentioned are occurring, there are usually present others of a less definite character. The patient loses flesh, he is restless and slightly feverish, the appetite is deficient, the bones and joints ache, and the hair becomes dry and thin. Now and then swellings occur on certain bones, more especially on those of the skull; but in this stage periostitis is always slight and transitory, and never leads to suppuration. Such are the various symptoms which make up what are called the secondary symptoms of Syphilis. In many cases only a few of them occur, the rash on the skin, and the ulcers in the tonsils being those which are most constant. In many individuals all the secondary symptoms have disappeared within six months of the original contagion; but in a larger number, a year elapses before such is the case. It is rare for any symptoms belonging to the secondary group to linger after eighteen months have passed, although their effects are often seen much later. That these symptoms will disappear in a most satisfactory manner, quite irrespective of any treatment which may be adopted, is a fact confirmed by every day's experience.

After the group of secondary symptoms has passed away, there usually follows a period of apparent health, during which the patient believes himself wholly cured.[1] After this interval, which may vary in length from a few months to many years, symptoms of a different kind ensue. Inflammatory indurations of a chronic character, and tending slowly to suppuration and ulceration, occur in the deep-seated tissues, or in the deeper parts of the superficial ones. In this way the skin, the subcutaneous tissue, the periosteum, the muscles, the internal viscera, the organs of special sense, and even the cerebro-spinal system itself may be attacked. A remarkable difference is to be observed between secondary and tertiary symptoms, as regards the symmetry of the inflammations produced. In the secondary stage, a tendency to symmetry, often very exact, is observed, proving that the producing cause is free in the blood, and is supplied impartially to both halves of the body. In the tertiary stage, the lesions are often single, or if multiple, they display but little accuracy as to symmetrical arrangement. This fact implies that they result from impaired organization of the solids, rather than from any poison still existing in the circulating fluids. The occurrence of the tertiary symptoms is to be explained by the fact, that during the exanthematic stage, when the whole blood was loaded with the virus, the various solids have received from that poisoned blood the elements necessary for their growth, and have been built up, so to speak, with syphilised plasma. Hence an impairment of organisation in such tissues, and a liability under slight exciting causes, or even in the ordinary course of nutritional change, to the occurrence of specific forms of inflammation. It is easy to see, that in Syphilis, with its very prolonged period of blood-poisoning, the risk of permanent tissue modification must be much greater than in the other fevers, in which it is so short. During the exanthematic stage of small-pox, probably but little in the way of nutrition is effected; the changes are chiefly those of waste and disintegration. From this, it follows that the more prolonged the secondary stage in Syphilis, the greater the risk of tertiary symptoms, and that severe febrile disturbance during this stage, by interfering with nutritional development, tends also in the same direction. Do we get from these considerations any glimpse into the principles of treatment? My impression is decided that tertiary symptoms, as a rule, follow earlier in the young than in the old, and if so, the fact may be explained by reference to the greater rapidity of elemental change in the former.

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[1] The interval which elapses between the well characterised secondary symptoms and the well characterised tertiary ones, is one of different degrees of immunity in different cases. In many, I think the majority, the poison is wholly latent, and the patient experiences nothing whatever to remind him of his taint. In many others, however, recurrences of symptoms, which it is difficult to assign to either group, continue to show themselves. Superficial sores on the tongue or the mucous membrane of the mouth, isolated patches of scaly or desquamating eruption on the skin, especially psoriasis palmaris, are the more frequent of these. Sometimes they are symmetrical; at others not so. Such symptoms may continue to recur for many years (even to twenty) after the contagion. They probably depend rather on permanent tissue contamination than on still existing blood disease. At any rate I may safely assert that we never witness any true recurrence of the secondary epoch. The eruption is rarely very copious, and rarely of such a character as to deceive an experienced eye; nor is it attended by the ulcers in the tonsils and iritis, which are so common in the secondary stage. In rare instances, at an interval of perhaps a year or eighteen months after infection, a relapse of a general symmetrical rash may occur. This rash may assume the form of rupia and be attended by severe constitutional symptoms.

An idea is prevalent that syphilitic iritis may occur at almost any period in the course of the disease. I speak after close attention to this subject, when I assert that all our well-marked examples of this disease present themselves amongst the secondary phenomena. From three to six months after the chancre is the usual date. When once iritis has occurred, and adhesions have been left, relapses are liable to happen, and it is this fact which has led to the error which I am endeavouring to confute. In these relapsing cases, however, the symptoms are very different from those of the first attack. The effusion of lymph is much less free, no nodules are seen, and rarely are both eyes simultaneously affected.

The following is a statement, in tabular form, of the various symptoms of the different stages of Syphilis.

1st Stage.—*Inoculation* and *Incubation*. Average duration, six weeks to two months.	An ulcer with indurated base and but very sparing secretion. Induration of the nearest lymphatic glands, with but little adjacent inflammation or tendency to suppurate. Slight febrile disturbance. Slight enlargement of lymphatic glands in all parts.
2d Stage. — *Exanthem*, or Secondary Symptoms. Average duration, two to six months; may extend over a year.	Symmetrical ulcers in the tonsils, not spreading either in width or depth. A symmetrical eruption on the skin. Condylomatous patches on the mucous surfaces, and on the skin adjacent to the mucous orifices, usually symmetrical. Iritis or retinitis; mostly symmetrical. Pains in bones and joints. Febrile disturbance. Loss of hair.
3d Stage.—Interval of Latency or of Relapses. May vary from a few months to many years.	In some cases the patient is wholly free from symptoms, but in a certain number reminders occur from time to time in the form of scattered scaly patches, psoriasis palmaris, sores on the tongue, lips, &c.
4th Stage.—Tertiary Symptoms or Sequelæ. Of uncertain duration, and characterized by a remarkable tendency to relapse.	Unsymmetrical ulcerations in the mouth and throat, tending to spread widely and deeply. Unsymmetrical ulcerations on the skin. Nodes of periosteum, cellular tissue, muscle, tendon, fascia, or nerve; not usually symmetrical, chronic in progress, tending to ulcerate or even to slough. Diseases of viscera.

Having thus sketched the normal course of syphilitic fever in its several stages and its sequelæ, we must next consider the conditions under which its orderly evolution may be interfered with. These conditions are precisely the same as those which disturb the course of any other exanthem. We have, 1st, idiosyncrasy; 2d, the co-existence of some diathesis or of some other specific disease; 3d, immunity, partial or complete, obtained by a previous attack of the same disease; 4th, inherited immunity, partial or complete; 5th, imperfect inoculation; 6th, the influence of treatment.

1st. To take first *idiosyncrasy*, I may simply remark that it is a matter of general experience that certain constitutions resist the specific animal poisons in a most remarkable manner. We are unable to offer any explanation of the fact; on the other hand we meet with those who succumb easily and suffer severely. The influence of these inexplicable peculiarities in individuals is frequently observed in reference to Syphilis.

2nd. The existence of some *special diathesis* or of some *other specific fever* at the time of syphilitic inoculation may modify the course of the latter. Possibly we overrate rather than otherwise the effects of these influences. Although it is not infrequent to find a delicate scrofulous subject suffering with unusual severity from syphilitic poisoning, yet the converse is almost equally common, in which either the delicate escape easily or the robust suffer very severely. There can be little doubt, however, that the tendency to suppuration and ulceration is much greater in those of fair skin and sanguine temperament than it is in others. The influence of diathesis (scrofula) is also often felt in preventing recourse to specific treatment. There is probably no reason to believe that the existence either of a diathesis or of another specific fever will modify the duration of the several stages of Syphilis.

3rd. *A previous attack of the same disease* is well known to exert a most important influence upon the course of a second in the same individual. It is generally understood that Syphilis, once had, is protective in the majority of cases against any second attack, and we have but little trustworthy clinical evidence as to the nature of second attacks when they do occur. There is reason to believe that second inoculations are common, and that they usually end in the production only of the soft chancre (abortive sore).

I have myself witnessed in one instance two attacks of Syphilis in the same patient, and in it the disease was slight on the second occasion. It appeared to have undergone quite as much modification as we usually observe in small-pox after successful and recent vaccination. A young surgeon consulted me for an indurated sore, in 1860. He was treated by mercury, but suffered very severely from all the usual train of secondary symptoms. It was two years before he had wholly got rid of the latter, and from that time to the present he has enjoyed excellent health. In April, 1865, he contracted another sore, which indurated, and was followed in a month by a copious roseolous rash. Excepting slight redness in the tonsils, there was no sore throat. He was scarcely ill, and both chancre and rash disappeared in a comparatively short time. I have seen other cases in which it seemed probable that the patient had had true Syphilis twice, but this is the only one in which both attacks have occurred under my own observation.

M. Diday holds that second attacks are not so rare as they have been thought, and records twenty-seven cases which have been under his own observation. Of these, however, in sixteen a chancre only occurred, indurated, it is true, but without secondary consequences. (Some of these may have been examples of the relapsing chancre, and not true instances of second contagion). In nine others the secondary symptoms were mild, and in two only were they severe. In these last two the average interval between the first and second attack was nineteen years and a half. (*See* New Syd. Soc. Year-book for 1862, p. 233.)

It is clear from these facts that second attacks of true Syphilis are very infrequent, and that when they occur within a moderate period of distance from the first, that the character of the disease is much modified. In these respects Syphilis conforms closely to all that is ascertained regarding the other exanthemata. An interesting fact in connexion with my own case of second attack of Syphilis (given above) is that the same patient had also had two well-marked but mild attacks of small-pox, the interval between the two having been four years.

4th. Of yet wider importance is the question as to *the influence of disease in the parent in affording protection, partial or complete, to the offspring.* If we grant, as we must, the two postulates—first, that Syphilis is transmissible to offspring, and second, that it is protective for a certain time against second contagion—then we are obliged to admit that just as the disease itself may be transmitted, so may the immunity which it affords. Here again we have as yet very little clinical evidence on which to build, but what we do possess certainly favours the view that those who have suffered severely in infancy from inherited disease are to some extent protected. In the history of Congenital Syphilis, however, nothing is more common than to meet with cases in which the eldest child of a family suffered severely in infancy, the second slightly, the third still more slightly, and the others not at all. I have at present several families under observation in which this has been the case, and in which all the children have lived, and the intervals between them are but short. The younger members of such families often appear to be in robust health. Now, if in such cases the oldest enjoys immunity, probably the second also does so in some degree, and so on through the whole, the degree of protection diminishing in ratio to the distance from the original taint. Do we not here touch upon a law of the utmost importance, not only in respect to Syphilis, but to its congeners also? Is it not probable that a very considerable portion of the community, being the descendants of those who have suffered, enjoy in a certain degree, infinitely slight in many but powerful in others, immunity from further attacks? The manner in which a slight degree of inherited immunity would become manifested would probably not be in entire escape from contagion, but in the production of a much milder form of the disease. This is what occurs in cases of small-pox after vaccination, or after a previous attack of the true disease, and indeed in second attacks of any of the specific fevers. It is surely impossible to believe that the constitution of a person who has passed through the stages of any of these diseases ever again returns into precisely the same condition in relation to the virus in question that it occupied before, and it is equally inconceivable but that some share of this peculiarity shall be transmitted to offspring. A child born of parents neither of whom are liable to small-pox or to Syphilis, as the case may be, must be in a different position, as regards those diseases, from the child of parents both of whom are liable. In like manner a half result ought to be expected where one parent is exempt and the other liable. Now it is a matter of well-proven observation that any specific disease will be especially severe when imported into a community previously free from it. The ravages of small-pox in a virgin race are something far beyond what is ever known in a community long accustomed to the disease. There are also good reasons for believing that Syphilis has become during the last two centuries a milder disease than it was when it first invaded Europe. This amelioration we may most satisfactorily explain by recourse to the hypothesis above suggested.[1]

5th. The next modifying influence which we have to examine is *imperfect contagion.* A large majority of the accidental inoculations by which Syphilis is conveyed are probably impure in a double sense. The essential virus is mixed with other fluids, is diluted, drowned, it may be, in common pus and other secretions. Then again the fluid merely comes in contact with a thin mucous surface; no precautions are taken to secure its gaining entrance into the tissue. Hence, as already explained, a majority of such contagions prove abortive. It is probable however that through all these difficulties the essential virus retains its characters unmodified, and that if it once produce its own first effects all the rest will follow. However small the quantity producing it, if once the sore have become indurated, the usual results may be expected. We have no reason for believing that there is any second variety of Constitutional Syphilis, other than that which follows an indurated sore. The questions as to imperfect contagion therefore concern the surgeon rather than the physician. Under its influence a great variety of venereal primary sores are produced, but we have no real variations in the specific fever and its results.[1]

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[1] This subject will be found very ably treated in Mr. Lee's Lectures on Syphilis (Lecture xi. page 209). Mr. Lee quotes the important observations of Dr. Ferguson (1812) as to the mildness of Syphilis amongst the Portuguese being explained by the acquisition of hereditary immunity, and adds, "That which Dr. Ferguson observed in his day may be seen at present. A person who has had hereditary Syphilis in his youth, will either not contract the infecting form of Syphilis in after life, or will have it in a modified form."

Four years ago I published in the British Medical Journal some cases in which patients who had suffered from inherited Syphilis subsequently contracted venereal sores. These cases were, I believe, the first facts relating to the subject which had been recorded. Others had arrived at the same conclusions, but it was by *à priori* reasoning rather than by deduction from facts.

Subsequently I published a case in which a patient who was the subject of inherited taint, not only contracted a venereal sore, but experienced an outbreak of constitutiona symptoms. This young man is still under my care, and suffers from inherited Syphili and acquired Syphilis at the same time. I have recorded a number of facts bearing on this subject in the second volume of the London Hospital Reports.

Lastly, we have to ask the all-important question, *whether the ordinary evolution of Syphilis can be altered in any way by measures of treatment.* It will probably be admitted that physicians have abandoned the idea that it is practicable by medication to regulate in any way the course of the other exanthemata. They are generally acknowledged to be diseases which always run their course. With the exception perhaps of cinchonism as a remedy for malarial fever, no single specific in the present day enjoys any repute as to cutting short the course of these diseases. But we must not too hastily assume *à priori* that the same will hold true as to Syphilis. It is possible that the stages of the other exanthemata are too short to permit of the beneficial influence of antidotes. Few questions as to therapeutics have been more hotly debated than the efficiency of certain drugs in reference to Syphilis. By some their specific power has been positively asserted, and by others as strenuously denied. As far as the purposes of our present argument are concerned, we may, I think, admit that there is no proof that the exanthematic stage of Syphilis can be prevented. If the sore have presented well-marked induration, a rash more or less copious is almost certain to follow in due time. A few exceptions occur, but they are as frequent when no treatment has been used as under opposite conditions. The statistics which have been collected on this point are for the most part valueless, because the kinds of primary sore have not been carefully distinguished. Any conclusions of trustworthy character must be based on the observation of indurated chancres only. For myself, I may state that I have treated some hundreds of these by the mercurial plan, and that in a considerable number of others I have carefully abstained from all medication, and that I am not in a position to record any single instance in which after mercurial treatment no exanthem followed.[2]

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[1] If we reflect on the mode in which syphilitic inoculation is usually effected, the wonder will be not that apparent varieties as to both primary and secondary symptoms occur, but that the disease preserves so close a connexion with its type as it undoubtedly does. Here, if anywhere, are the conditions under which we might expect a new species to originate. In the first place the virus is constantly mixed with other secretions, and very frequently with those of inflammatory origin. In a great many instances the person from whom the contagion is received is one whose own body has been previously rendered proof against the disease. Most prostitutes probably suffer from Syphilis early in life, and during the greater part of the period during which they continue their vocation are incapable of being themselves again affected by true Syphilis, although still liable to contract and to transmit primary sores of a modified character. Then not only must we make allowance for differences in the kind of secretion with which the inoculation is effected, but also for differences in the recipient's state as regards it. Hence the differences in the type of cutaneous rash which follows; from a roseola to psoriasis and to rupia.

[2] By this I mean that I have never seen a case in which after a well indurated sore I kept the patient continuously under observation, and assured myself that he never had any constitutional symptoms. I have seen many in which the constitutional symptoms

That mercury can procure the healing of syphilitic sores and the absorption of syphilitic lymph, no one who has had opportunities of observation, and who dare credit the evidence of his senses, can doubt; but that it can prevent the occurrence of one of the stages of the disease, is a very different assertion. I shall discuss the question of treatment in its practical aspects at a further part of this essay; for the present, and in reference merely to the natural history of the disease, it may suffice to observe that there is no more proof that it can prevent the evolution of the exanthem of Syphilis, than that it can do the same in variola. Whether the tertiary symptoms can be prevented or made milder by treatment is again another question, since they, strictly speaking, do not constitute a true stage, but are rather the sequel, more or less accidental, of the secondary one. Any remedy which, although impotent to prevent, is yet able to modify and shorten the secondary stage, may very possibly influence the occurrence or otherwise of the tertiary inflammations; and whether mercury does so or not, must be determined solely on clinical evidence.

Modes of Communication.—Whilst the other exanthemata are for the most part communicable only by direct contagion or infection to the individual concerned, Syphilis, in consequence of its very protracted duration, may be conveyed in any one of three different modes. First, contagion direct to the individual; second, contagion indirect through the fœtus (possible only in women); and third, by hereditary transmission.

The period during which direct contagion is possible extends from the first appearance of an indurated chancre to the decline of the exanthem. The primary sore is more actively contagious than are any in the secondary stage, but there can be no doubt that under favourable conditions the germs of the disease may be conveyed by the latter. When Syphilis is communicated to a mother by contamination from the fluids of a fœtus with which she is pregnant, the course of the disease is materially different from what it is when received by other means. The absorbed materies seems to be scarcely capable of breeding in the blood of its recipient; it merely contaminates it, the degree of the contamination being in exact proportion to the amount received. The evidence of contamination is greatest during the pregnancy, and increases with each successive one. The symptoms produced are of the tertiary class only; for the most part the secondary stage is wholly omitted. A taint thus obtained rarely attains any high degree of severity.

When Syphilis is transmitted from parent to offspring, various important peculiarities are observed in its manifestations. In the first place the phenomena of the secondary and tertiary stages not very unfrequently occur together, or at any rate we have a superficial rash

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were so slight that they might easily have been overlooked, but these are sufficiently frequent without treatment to make us very cautious in assuming, when such a result follows mercurial treatment, that it is a *propter hoc.* Many patients whom I have treated by mercury for the chancre have been lost sight of as soon as the latter was healed, and it is of course possible that in some of these no secondary stage occurred.

on the skin resembling a secondary one, coincident with nodes and with deposits in the viscera. These cases are however exceptional, and as a rule the stages occur as in the adult, the secondary rash disappearing after a few months, and there being a prolonged period of health before the tertiary symptoms show themselves. A few symptoms are peculiar to the inherited disease, and do not occur in adults who have acquired it. Amongst these I may mention, of the secondary stage, diffuse stomatitis without ulcers, diffuse inflammation of the mucous membrane, of the nares resulting in the well-known symptom of snuffles, and of the tertiary ones a form of phagedænic lupus and interstitial inflammation of the cornea. The latter, which is a common and very well marked condition in inherited Syphilis, has no parallel condition whatever in the acquired disease. Deafness and amaurosis from nerve or cerebral disease are both of them far more common in the inherited form of the disease than they are in that which is acquired. The effect of the syphilitic poison upon the ovum is in many instances to destroy its vitality at an early period, and consequently to induce abortion. Unfortunately this is far from being its constant effect. In the great majority of such conceptions the tainted fœtus is carried to its full period. In exceptional instances it is then brought into the world with manifestations of its disease apparent in the form of skin disease; but in most this is not so, and the infants who when a few weeks old will suffer most severely appear at first to be perfectly healthy. In these a period of from a fortnight to two months usually elapses, and then simultaneously a rash appears, and the nostrils become stopped by swelling. At this stage the mouth is usually hot, its mucous membrane red and tumid, and the gums swollen. The child wastes, and assumes a shrivelled senile aspect. Sometimes acute, well characterised iritis occurs. Condylomata are frequently seen. The cutaneous exanthem may vary in character, much as we find it does in the adult. Many children die during this evolution of secondary symptoms. If they survive they usually in the course of a year get rid of all traces of disease, excepting perhaps an unusual pallor of skin, and certain scars which may have been left in the face by the eruption, and an expanded nasal bridge caused by the long-continued swelling of the parts within.

I have said above that the tertiary and secondary stages are sometimes strangely mixed in the early symptoms presented by syphilitic infants. Amongst those which we occasionally meet with under these circumstances are nodes of the long bones, nodes of cellular tissue, of tendon, or of muscle, and disease of the liver, kidneys, thymus gland, &c. Such children are certainly more liable than others to serous inflammations. Serous arachnitis to a slight extent is very common, and pleurisy is not an infrequent cause of death.

A condition of extreme anæmia usually results during the outbreak of early symptoms in a syphilitic infant, and from this death often results. In many cases however the child does not emaciate, but retains an appearance of good health which is remarkable, considering the nature of the disease. I have occasionally seen an infant who was well grown, stout and strong in an unusual degree, who yet presented well characterised indications of inherited taint.

In the child as in the adult the secondary symptoms pass away in due time, and a period of health or latency ensues, of variable duration, after which the tertiary phenomena show themselves. These are of precisely the same character as in the adult, with however the addition of several others which are not met with in connexion with the acquired disease. There are few more remarkable facts in the history of this most interesting malady than that the disease known as Interstitial, or according to Mr. Dixon as Syphilitic Keratitis, should never occur as a consequence of acquired disease, but only in the inherited form. I must also here note a remarkable exception to what I have stated to be the characteristic of tertiary symptoms in the adult, that they are exceptionally symmetrical. This form of keratitis, although it often occurs many years after the secondary stage, is as a rule symmetrical. So also are the nerve affections, which result in the forms of deafness and amaurosis which we now and then encounter in these patients. As a rule, I believe all syphilitic symptoms in the inherited disease, without regard to stage, are symmetrical. In the cases in which tertiary and secondary symptoms in infants appear to occur together, the latter are rarely well characterised. Thus I do not know of any instance in which a copious scaly or papular rash with acute iritis were coincident with any symptom of a tertiary kind. It has been asserted that a parent transmits to his child the precise form of Syphilis from which he at the time suffers. But to any rule of this kind exceptions are far more frequent than are confirmatory instances. It is very common for a man who does not himself display a single symptom of any kind, and who appears to be in perfect health, to beget a syphilitic child, the symptoms displayed by the child being usually those of the secondary class. There is no doubt that the nearer to the occurrence of the primary symptoms in the parent is the birth of the offspring, the more certain is the latter to show symptoms of a severe character, and typically secondary in stage. Instances, however, are met with in which infants, born ten years after the original disease in the parent, still display first a secondary rash, with the characteristic snuffles, &c. In several instances I have known a whole family of children, born during a period of from five to ten years, display each one the characteristic and transitory rash soon after birth.[1]

The following appear to me to be well-established conclusions as to the transmission of inherited taint.

1st.—In all stages of Constitutional Syphilis—whether during the secondary or tertiary symptoms, or even during a protracted period of latency—an individual may become the parent of a tainted child.

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[1] For facts on this subject I may refer the reader to my paper in the London Hospital Reports, vol. ii. p. 184, *et seq.*

The degree of severity of the inherited taint will be in proportion to the shortness of the period which has elapsed.

2nd.—A child may inherit Syphilis in a severe form from but one parent—from its father alone, or from its mother alone.

3rd.—When both parents are the subjects of Syphilis a child is more certain to suffer, and also more likely to suffer severely, than when only one is so.

4th.—We have as yet no data on which to ground an opinion as to whether a child is more likely to suffer severely when its father is the source of contamination than when it derives the disease from its mother, or the reverse.

5th.—In a large proportion of the cases met with in practice, the taint is derived from the father only.

In connexion with the hereditary transmission of Syphilis, an exceedingly important question arises as to whether any degree of taint is transmissible to the third generation. There is no doubt that persons of marriageable age often present heredito-syphilitic lesions in an active stage, such as keratitis and nodes. I have repeatedly seen patients of various ages, from twenty to eight-and-twenty, become the subjects of syphilitic keratitis for the first time. We might conjecture that such persons would be likely to transmit to their offspring some degree of taint, seeing that the taint is still in full activity in their own bodies. I am not aware that any facts have as yet been published on this question. Conjectures abound, and several surgeons have expressed their belief, that the influence of Syphilis once acquired is felt through several subsequent generations. About eight cases have come under my own observation in which persons, undoubtedly the subjects of inherited disease, have become parents. With one exception, I have never been able to discover any evidence of disease in the offspring. In several instances the offspring appeared to be in excellent health. I have always made a point of seeing the children for myself, never relying upon the parents' statement—a precaution which is essential, as I have here occasion to illustrate. The exceptional case just alluded to is strongly in favour of the belief that the third generation may suffer. As no parallel one is on record, I think its details worthy of brief mention. A respectable young woman came to me about six months ago on account of an inflamed eye. She had interstitial keratitis in a typical form, her teeth were notched, and her physiognomy characteristic. She told me that she was suckling her first child, an infant of two months. I inquired if it were healthy. She said it was a fine baby and ailed nothing whatever. I asked her to bring it with her at her next visit. She did so, and on having it stripped I found it covered with coppery blotches, with condylomata at the anus, and snuffles in the nose. Under subsequent treatment by mercury all these symptoms disappeared. There remains of course the source of fallacy that this child's parents, one or other of them, may have had acquired Syphilis. As to its father, I may state that he has been long under my treatment for sycosis, and that I have made the most detailed inquiry of him as to any venereal disease. I believe strongly that he has never had any. A fact, which is perhaps of more value than his own statement, is, that his sycosis has not been in the least benefited by iodide of potassium. Of course I have not ventured to insult him by inquiring as to his wife's antecedents, but there is no reason to entertain suspicion in that quarter, whilst the fact that she is the subject of inherited disease makes it probable that she would not be liable to the acquired disease. Having therefore carefully balanced the evidence, I incline to believe that we have in this instance an example of the transmission of Syphilis to the third generation.

TERTIARY SYMPTOMS OR SEQUELÆ.—I have endeavoured to draw a strong line of distinction between secondary and tertiary symptoms. The secondary phenomena constitute a stage; they come on at a certain known period; they are in their nature transitory, and undergo spontaneous cure; they affect the two halves of the body at the same time, proving that they depend upon blood-poisoning; when once passed they rarely return. The tertiary symptoms are not so properly a stage, but must count rather as the sequelæ, more or less accidental, of the preceding stages. They are as a rule not symmetrical, making it seem improbable that they depend upon blood-taint; they have no tendency to spontaneous cure—quite the reverse. They relapse over and over again after remedial treatment. The period which intervenes before their outbreak is of very different length in different cases, and in many they never occur at all. From these facts we infer that they are due rather to the ill constitution of the affected structures than to any free virus still circulating in the blood. Let us briefly enumerate the principal tertiary symptoms which occur in acquired Syphilis.

We may conveniently take them in their relation to special organs or structures. *First, the skin and mucous membranes.* Tertiary affections of these tissues differ in a most marked manner from those which occur in the secondary stage. With the exception perhaps of palmar psoriasis, they all involve ulceration of greater or less depth, and consequently leave cicatrices. Very frequently the patch assumes a crescentic form, spreading at its edges and healing in its centre the well-known horse-shoe or serpiginous ulcer. If the disease commence in the middle line it may spread equally on the two sides, and may thus appear to be symmetrical; but it is decidedly unusual for symmetrically-placed patches to appear on the opposite limbs or on corresponding parts of the trunk. In many cases the skin is involved secondarily to the sub-cutaneous cellular tissue, the disease having begun as a gummous tumour or node of the cellular tissue. A form of lupus attended by rapidly-spreading phagedænic ulceration, occasionally occurs in tertiary syphilis, but there is good reason for believing that the common forms of lupus, whether exedens or non-exedens, have no connexion whatever with syphilitic taint.

The appendages of the skin, the nails and hair, are frequently affected during the secondary stage, and but very rarely at later periods.

The most frequent affection of the mucous membranes which we encounter in connexion with tertiary syphilis, is a rapidly-spreading ulceration of the palate and pharynx. This again is totally different from the throat affections which occur in the earlier stages. Instead of being superficial and marked chiefly by swelling and inflammatory deposit, it is characterised by deep ulceration and loss of tissue. Instead of showing itself symmetrically on the two sides, it commences at one, two, or more points, and spreads quite irregularly. The cicatrices left by these deep ulcerations not infrequently narrow the pharynx and occasion difficulty in deglutition. In a few cases the ulceration may extend down the œsophagus, and in many the larynx is involved. Every now and then we see cases of tertiary syphilitic ulceration of the mucous membrane of the rectum, and again we must note that it is ulceration, and that it is not attended by the development of condylomata or mucous patches, as usually seen in secondary Syphilis. Stricture of the rectum is much to be feared when these ulcerations heal. Several authors have described cases resembling dysentery in all their symptoms, but occurring in syphilitic patients, and cured by anti-syphilitic remedies. Mr. Paget has recently recorded a case of this kind, and I have myself seen some very well-marked ones. It is probable that in such cases ulceration of the mucous membrane at a considerable distance above the anus is present. I have seen several cases in which syphilitic ulceration extended higher than the finger could reach.

The cellular tissue is frequently involved in common with muscle, with periosteum, or with fascia. In not a few cases, however, we meet with what are called cellular nodes, in which the disease begins, and is, up to a certain period, confined to this tissue.

These may occur in any part of the body, but are much more usually met with in the lower extremities than in any other part. They are very common close to the knee, and especially so in the female sex. It is a very interesting fact in respect to these cellular nodes that they are comparatively very infrequent in men. Whether this is to be explained by the greater abundance of cellular tissue in women, or by the fact that many women obtain Syphilis in a manner wholly peculiar to them, that is, by fœtal contagion, may be open to some question; probably both influences have their share in the result.

In the early stage of a cellular node we find a small lump of induration, often exceedingly tender. At first it is firm, but as it extends it becomes doughy and softer. When of considerable size there is frequently a very deceptive sense of fluctuation in it. The overlying skin becomes adherent and of a dusky red colour. At length ulceration takes place, and a large core is exposed, consisting of sodden and infiltrated tissues, much resembling in appearance soaked wash-leather. Unless specific remedies are used, this core is very slow in separating, and the ulceration of the skin over it may spread widely.

Cellular nodes are not infrequently multiple, but more usually single. The patient frequently has scars of former ones on the opposite limb, but it is exceptional to find them simultaneously present on corresponding parts.

A period varying from four to ten or fifteen years has usually elapsed between the occurrence of primary contagion and the development of cellular nodes. In close connexion with syphilitic inflammation of the cellular tissue, we must mention that of *the subcutaneous bursa.* It is not at all uncommon for a bursa to suffer in connexion with the disease of the tissue around it, and sometimes there appears to be clear evidence that the disease began in the bursa itself. The bursa in front of the patella is the one most frequently involved.

When ulceration takes place the inflamed bursa is usually involved in the core, and has to be entirely removed before healing can ensue.

Inflammations of the periosteum and bones have for long occupied the most prominent place amongst the tertiary symptoms of Syphilis, and they are still some of the most common. In enumerating the symptoms which characterise the secondary stage, we have mentioned pains in the bones, attended occasionally by slight and temporary swelling. This kind of periostitis, however, never lasts long, and as far as my own observation goes, never leads to suppuration. True nodes seldom occur until at least two years have passed since the first contagion, and generally the period is much longer. They may affect almost any parts of the osseous system, but the bones which are superficial, and therefore most exposed to external influences, are those most frequently attacked; *e.g.* the calvaria, tibiæ, and the clavicles.

The bones of the palate, the alveolar processes of the maxillæ, the vomer, and other bones in the nasal passages, are very frequently affected, and when such is the case, exfoliation of portions usually occurs.

Syphilitic periostitis may vary considerably in its degree of severity and in its tendencies.

In some cases there is but little of acute inflammation, and the result is a great thickening of the bone affected, without the occurrence of suppuration. This frequently occurs in the bones of the skull—the whole calvaria acquiring greatly increased thickness and density. It is also not uncommon on the surface of the tibia and other long bones, constituting what is known as the osseous node. In other cases suppuration occurs, and in these very frequently large portions of cellular tissue become involved, and we have a swelling consisting in part of a periosteal abscess and in part of a cellular node. When the bone is exposed by ulceration, exfoliation of portions often results.

When the bones of the skull are attacked by syphilitic periostitis it is very possible that inflammation may occur internally as well as superficially, and that we may have symptoms referable either to irritation of the cerebral coverings or to compression consequent upon intra-cranial abscess. In association with nodes on the skull, various symptoms of mental disturbance show themselves; extreme irritability of temper, liability to fits of uncontrollable passion, melancholia, and sometimes acute mania occur. These symptoms of mental disturbance may or may not be associated with those of local paralysis. They not infrequently result in attempts at suicide. The proof that they really are dependent on syphilitic lesions is afforded by the ease and rapidity with which they are relieved by the iodide of potassium. Some remarkable instances of this kind have recently been under my care.

Periosteal nodes are not very frequently met with on the short bones; we must however be prepared to recognise them occasionally on these also. The patella and the os calcis are not very infrequently affected, and now and then the other bones of the tarsus or carpus suffer.

Diseases of the muscular system occur chiefly amongst the most remote sequelæ of Syphilis, and they are by no means frequent. They usually take the form of nodes or gummata, developed in the substance of some single muscle. The induration is usually very considerable, and in many parts abruptly limited. The diagnosis from cancer is often very difficult, and many a mistake leading to an unnecessary operation and to a supposed permanent cure of cancer has occurred.

The muscular substance of the tongue is that most frequently attacked, but they have been met with in almost all the muscles of the body. We may mention especially the sterno-mastoid, the masseter, the supra- and infra-spinati, the gastrocnemius and the rectus femoris.

I have recently had under care an extremely interesting case in which a tumour, which we at first suspected to be cancer, was developed in the left masseter of a lady who had twenty years before suffered from Syphilis. She presented at the time the tumour appeared no other syphilitic symptoms, and the correct history was only obtained with much difficulty. The tumour has wholly disappeared under the use of the iodide of potassium.

Some forms of syphilitic indurations of the tongue are exceedingly difficult to distinguish from cancer. They are very hard, have well-defined edges, are painful, and when they ulcerate present an unhealthy surface. Iodide of potassium in full doses will usually in the course of a week or ten days clear up the diagnosis. The heart itself is sometimes the seat of syphilitic nodes. Of this, M. Ricord[1] was, I believe, the first to record an example; but several others have been subsequently mentioned by other observers.

The Glandular System.—The chronic enlargements of the lymphatic glands, sometimes resulting in suppuration, are every now and then met with as the sequelæ of syphilis, but it does not appear to me that they occupy any very important position. It is a remarkable fact in reference to tertiary syphilitic lesions generally, that they do not cause any secondary enlargement of the adjacent lymphatic glands. This is true of syphilitic ulcerations of the skin and mucous membranes, of all the various forms of node, and of syphilitic tumours in muscles, and it often constitutes a very useful means of differential diagnosis between cancer and syphilis.

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[1] See Traité complete des Maladies Vénériennes, Planche xxix. In this instance the patient was a man aged 41, who had suffered from a chancre followed by constitutional symptoms eleven years prior to his death.

The Internal Viscera.—Of late years the investigations of pathologists have fully confirmed the conjectures of the older writers on Syphilis, as to the frequency with which the viscera of the trunk, and more especially the liver, suffer in constitutional syphilis. In connexion with this subject we must especially mention the very valuable contributions of Dr. Wilks. As to the exact period in the course of the disease at which the viscera are attacked, it is difficult to obtain any positive evidence. What we discover in the post mortem examination is usually the result of long past disease, and it is comparatively infrequent to find it in a recent stage. What evidence we have, however, favours the belief that it is not until the later periods that the viscera suffer.

The liver appears to be far more frequently affected than any other organ. Indeed, in the examination of the bodies of those who have suffered from tertiary syphilis, it is decidedly exceptional not to find proof of hepatic mischief. The most common condition consists in large white patches of fibroid thickening on the surface of the organ. These patches are evidently cicatrical. The liver is knotted and puckered up by them, and bands of cicatrix dip from the surface into the substance of the organ. Sometimes, when the destruction has been great, the whole bulk of the organ is diminished. In recent disease the affected parts of the organ are enlarged, and on section exude a material not unlike bees-wax, or glutinous and gummy. I am not aware that abscesses have as yet been met with in the liver in supposed connexion with Syphilis. Virchow recognises two forms of disease—a capsular hepatitis and an interstitial hepatitis. Of these the capsular inflammation is the more common and the less serious. It is probable that the two are generally associated to a greater or less extent. Ascites occurs every now and then in connexion with syphilitic disease of the liver. An instance of it in a woman, the subject of inherited syphilis with a contracted liver, has recently been under my care. The disease was of several years' standing, and paracentesis had been repeatedly performed. By a long course of iodide of potassium, with ammonia, the fluid was entirely removed and her health much benefited. Ascites from liver disease is not very infrequent in the subjects of inherited taint.

Testes.—Syphilitic sarcocele or syphilitic orchitis has usually been classed by authors as a secondary symptom. I feel sure, however, that this is not quite correct. It is amongst the earlier of the sequelæ, but seldom if ever occurs during the secondary stage. It is commonly met with in conjunction with nodes, and with deep ulceration of the skin rather than with the superficial rash of the secondary epoch. It consists of the free effusion of lymph (fibro-plastic material) into the substance of the testis, or, more rarely, into the epididymis.

The swelling often attains a very considerable size, and when it does so it presents the peculiar feature of feeling very light in the hand. Syphilitic sarcocele is much more frequently symmetrical than any other form of tertiary syphilis. This circumstance we might expect from the fact that it occurs much nearer to the secondary stage than do most of the others.[1] Still, however, it is only exceptionally symmetrical.

Nervous System.—We come lastly to syphilitic affections of the nervous system itself.

I have previously adverted to the occasional occurrence of cerebral symptoms in connexion with syphilitic inflammation of the bones of the skull, and to the formation of intra-cranial nodes; but, quite apart from disease of its osseous case, the brain itself may suffer directly from the formation of tertiary syphilitic deposits in its structure. We may also have deposits of like nature into the substance of nerve trunks, producing special forms of local paralysis. To these isolated deposits the term syphilitic neuromata has been given, and several well-authenticated cases are on record in which the diagnosis has been confirmed by an autopsy. In a far greater number of cases the diagnosis has received an almost equally valuable confirmation in the cure of the disease by iodide of potassium. So frequently indeed is tertiary syphilis the cause of paralysis, that investigations in this direction ought never to be omitted in cases in which the nature of the disease is in the least doubtful. It is, indeed, safe to go further than this and to say that in all cases of paralysis, without evident cause, and in which syphilitic antecedents are even possible, it is advisable to try the effect of iodide of potassium. I allude chiefly to cases of paralysis of the cranial nerves, for it would appear that neuromata of these are more frequent than of the spinal ones. Of the cases of paralysis of the fifth nerve, of the third, fourth, and sixth, which have come under my notice at the Ophthalmic Hospital, a large proportion of these have been of syphilitic origin, and most of these have been cured by the administration of iodide of potassium.

Syphilitic affections of the nervous system are usually among the late tertiary phenomena. I have rarely seen them at an earlier period than about five years after the primary disease, and in most instances the interval is much longer. In many cases the patients have had time to regain the appearance of good health, and almost to forget the malady from which they had formerly suffered. Under such circumstances the diagnosis is often surrounded with difficulty.

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[1] On this point Mr. Curling writes, "Sir A. Cooper thinks that in the majority of cases, the disease attacks both testicles. The eight examples recorded in his work do not however bear out this remark, for in only two of them does it appear that both organs were attacked. According to my observation, the disease is more commonly confined to a single gland, though it occasionally affects both; and this also appears to be the opinion of Ricord."

Syphilitic affections of the nerves of special sense do not appear to be common in connexion with acquired disease, but they are not infrequent as results of inherited taint. In the subjects of the latter a form of cerebral deafness is often met with, and also one of complete blindness in association with white atrophy of the optic nerves.

TREATMENT OF SYPHILIS.—In approaching the question of the treatment of Syphilis we must always keep clearly in mind the facts which have been established as to its nature. Not indeed that *à priori* reasoning is to supersede empirical experience in such a matter, but rather that we shall do well to guide the one by the other. Viewing Syphilis as a disease of the zymotic class, caused by a specific virus, which accomplishes its development within the body of the infected person, and passes through distinct phases or stages, nothing can be more probable than that in order to influence its course we shall require very different measures in the different stages. The treatment which we should adopt in the onset of variola is not that which we should resort to after the exanthem has disappeared. We have then in respect to Syphilis to ask: First, what treatment should be adopted in the exanthematic or secondary stage. Second, what should be used against the very various sequelæ classed as tertiary symptoms. Third, whether there is any reason for believing that the development of stages, more especially of the exanthem, can be influenced by internal treatment adopted immediately after inoculation? Fourth, whether the sequelæ are rendered less or more severe by interference with the development of the early stages of the complaint.

As the present essay is on the *medical* aspects of Syphilis, we may suitably leave out of debate a matter of the utmost interest to the surgeon, that, namely, as to the prevention of constitutional infection by local treatment of the inoculated part (so-called "abortive treatment").

To cite an array of facts on this extensive subject would be wearisome, and without adequate result, nor should we more easily accomplish a summary of the very diverse opinions which have been published by medical authorities. The question as to the treatment of Syphilis resolves itself chiefly into one as to the efficiency or otherwise of mercury. We will submit for consideration answers to the following questions. Does mercury in any way influence the course of syphilitic symptoms? On this point I think almost all are unanimous. When given during the stage of induration of a chancre, mercury causes the absorption of the induration, and the healing of the sore; when given during the outbreak of the secondary rash, it causes the rash quickly to disappear; it also causes the ulcers in the tonsils to heal. In cases of iritis and retinitis we have the most conclusive proof of the rapid absorption of syphilitic lymph under mercurial influence, inasmuch as in each of these conditions the inflamed structure is directly under our inspection. If we inquire as to the value of mercury against the tertiary symptoms, we obtain a much less positive answer, but we shall still meet with evidence in proof that over many forms it possesses a most decided power.

Having seen that mercury does, beyond all doubt, possess the power of shortening the duration of the primary sore, or if not used until the secondary manifestations have appeared, of causing these latter to disappear; we next have to ask whether the mercurial cure of any single stage, whether primary or secondary, influences beneficially the subsequent progress of the disease. The difference between an antidote for the syphilitic virus and a remedy for extant syphilitic inflammations, must be clearly recognised. It is one which has been acknowledged from the time of Hunter to the present day. It appears to me that the balance of evidence is in favour of the belief that mercury is a most potent remedy against syphilitic inflammations, but that it does not act as an antidote to the virus. I fear we have but little proof that mercury tends, on the whole, to abridge the duration or mitigate the severity of the syphilitic fever and its sequelæ.

During about two years in my practice at the Metropolitan Free Hospital, I systematically abstained from adopting any treatment in my cases of indurated chancre and its consequences. The chancre and the rash were allowed to develop themselves and to disappear spontaneously, and they did so in a fairly satisfactory manner. The duration of each was considerably longer than when mercury is given; otherwise I could observe no difference. The rash did not appear earlier, nor was it more copious than in the cases in which the remedy had been used. On the whole I had no reason to think that the patients suffered from the experiment beyond the fact of a more prolonged illness. As to what may be the relative frequency of tertiary symptoms in these cases it is as yet too early to speak. I have also, in private practice, not infrequently treated indurated chancres and secondary rashes without giving mercury.[1]

As already stated, I think there is no proof whatever that by giving mercury for the primary sore, we diminish the probability that secondary symptoms will occur. These latter are for the most part inevitable, whatever may be the treatment employed. They are sometimes very slight indeed, and in some cases, perhaps, wholly omitted, but their non-occurrence is quite as frequent when mercury has not been given as under the opposite conditions. It is, therefore, unsafe to assume because in any one case in which mercury was given early, and no secondary symptoms ensued, that, therefore, the treatment prevented them. Such sequences are probably mere coincidences. Thousands of cases might be quoted in proof that mercurial absorption of the chancre does not prevent the secondary stage; and, further, that the mercurial treatment of both primary and secondary stages does not prevent the occurrence of tertiary sequelæ. We might also quote another class of facts in proof that mercury is not in any strict sense an antidote, those, namely, in which relapses occur either during or immediately after its use. These cases must be familiar to all. A patient, whilst actually salivated on account of iritis in one eye, becomes affected by the same inflammation in an acute form in the other, or just after the mercurial cure of iritis, retinitis occurs. Of the latter occurrence I have seen several marked examples. I admit that these quick relapses are exceptional, but they are still sufficiently common to become of great value in reference to the question under debate. The belief that mercury given in the early stages in any way complicates the case or adds to its subsequent severity is, I think, to a very large extent an error; at any rate it is quite certain that the worst forms of syphilitic symptoms, whether secondary or tertiary, not infrequently occur in cases in which no mercury has been used; especially is this a fact as regards tertiary symptoms, such, for instance, as extensive disease of the bones. In former days, when mercury was given so freely, it was not easy to find cases of tertiary syphilis without the history of previous mercurial treatment; in the present day, however, it is not at all infrequent.

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[1] I have preferred to speak from my own experience rather than to refer to the large amount of published evidence which exists. My own trial of the non-mercurial plan was made purely as an experiment, and without the slightest sentiment of partisanship.

If I might be allowed to express my own impression, founded as it is on a considerable number of facts, but for obvious reasons not easily susceptible of categorical proof, it would be to the effect that the course of Syphilis is on the whole rendered somewhat milder by early mercurial treatment.

Quite apart from the question as to the general influence of mercury upon the course of Syphilis, we must estimate its value in the speedy removal of local inflammations. We have ventured to consider that it is a proven and admitted fact that this remedy does produce the rapid absorption of syphilitic lymph. When this lymph is effused into the skin, or at the base of the original chancre, it may be a matter of little or no consequence whether it is allowed to remain two weeks or two months. There is no material danger as to the integrity of the organ concerned. In the case of the eye and certain other organs, however, the facts are very different. If the iritis be allowed to proceed unchecked, it will in all probability end in obliteration of the pupil, either partial or complete. It will effect but little to use atropine, unless we use mercury also, for in many cases during the acute stage of the inflammation the pupil can be scarcely made to dilate until the lymph effused into the iris is in part absorbed. The longer the lymph is allowed to remain, the longer the inflammatory process is allowed to continue unchecked, the greater will be the risk of disorganization of the structure implicated.

These remarks apply with yet more force to syphilitic retinitis than to iritis. That it is the bounden duty of the surgeon to administer specific remedies in these diseases, no one who has considered the facts can, I think, doubt. It is not uncommon to see the retina in a case of severe retinitis become almost clear after a fortnight's mercurial treatment, with corresponding benefit to the patient's vision. It is on the other hand very common to see this disease remain unchanged for several months, if mercurial treatment be not adopted.

Those anti-mercurialists who carry their doctrines so far as to refuse to employ specific remedies when the eye is attacked, incur a responsibility probably far greater than they suppose. A case has recently come under my own observation so much to the point that I must mention it.

A gentleman engaged in the city consulted me concerning some symptoms which I easily recognised as the sequelæ of syphilis. Amongst others he had muscæ and evidences of a past attack of retinitis. He gave me the history that he had been treated for the primary disease by a surgeon well known as an opponent to mercury. This gentleman explained to him in very strong terms the evils which he supposed to result from that drug, and so far secured his confidence that he continued under expectant treatment for several months. The eyes were attacked, and still specific remedies were abjured. "At length," said my patient, "when I was all but blind, and when for several weeks no improvement had occurred, I determined to take other advice, and consulted Mr. Critchett. Mr. Critchett assured me that the fear of mercury was all nonsense, and that the only chance for my sight was at once to go home, keep myself in a warm room, and take mercury till the mouth was sore. This I determined to do, and the result was, that in the course of a week, I could see very much better, and that subsequently I regained almost perfect sight."

In this case, not only did the mercurial treatment rapidly cure the retinitis, but it removed the syphilitic rash and restored the patient's general health in a way which, to him, was marvellous.

When syphilis attacks the larynx it becomes also of great consequence to adopt energetic treatment, on account of the danger to life which may accompany the local disease.

Although in the case of the skin we have to deal with an organ not essential to life, and the functions of which may be long interrupted with comparative impunity, yet it is still probable that a very extensive cutaneous inflammation, such as occurs in the exanthem of syphilis, is not wholly without its injurious influence on the general health; thus there may be some reason alleged for preferring to get rid of a secondary rash in a few weeks by mercury, rather than to allow it to disappear spontaneously after several months duration.

We come, lastly, to the question as to the treatment of the tertiary symptoms, or the sequelæ which occur several years after the contagion.

The marvellous power of the iodide of potassium in the cure of these affections is universally admitted, and has led to the almost entire disuse of mercury in their treatment. The only drawback to its employment is, that the cures thus effected are rarely permanent, and that relapses are very frequent in a short time after its suspension. Some surgeons of large experience hold that mercury, even in regard to the tertiary symptoms, is more efficient than the iodide in bringing about a permanent cure. This is, however, open to much doubt. Practically, in all cachectic subjects suffering from tertiary symptoms, we usually administer the iodide alone, and if the cachexia be less severe we combine it with a mercurial. Another general rule on this point is, that the earlier in the rôle of tertiary symptoms the greater the desirability of mercury, and the more remote from the original taint the more likely is the iodide alone to be found efficient. Thus, cases of node, of gummous tumour, of tumours in muscles, and of affections of the nervous system, all of which are among the later of the tertiary class, are usually treated most satisfactorily by iodide of potassium.

What little experience I have had of the treatment of tertiary syphilis, either by the mineral acids or by sarsaparilla, has not been favourable to these remedies. We must not overlook in the management of cases of syphilitic sequelæ, the importance of change of air, of nutritious diet, and general attention to the patient's health. Such sequelæ are especially apt to occur and to become severe in patients whose general health has been broken down. Any debilitating influence brought to bear upon a patient the subject of a latent taint may permit such taint to become active.

Thus, patients who have for many years enjoyed excellent health, and have believed themselves long ago and completely cured, may become, at the climacteric period, or when by any chance under-fed and enfeebled, the subjects of tertiary syphilis. Yet, whilst fully admitting the importance of sustaining the general vigour as a prophylactic measure against tertiary syphilis, we must keep in mind that these measures will by no means prove always efficient. Many of the worst cases of tertiary syphilis, whether consequent on inherited or acquired taint, occur in those whose circumstances of life enable them to enjoy every hygienic advantage.

With regard to the details of mercurial treatment, differences ot opinion still prevail amongst surgeons. The majority still think the stomach the most fitting organ by which to introduce the remedy into the blood. There is no doubt that absorption by the skin, whether by inunction or the moist vapour bath, is very efficient, but there is no proof that it is superior to the other. Under the able advocacy of Mr. Langston Parker, Mr. Lee, and others, the calomel vapour bath has of late years obtained much repute. I have often used it, and have still more frequently seen cases in which it has been used by others, and have not been able to persuade myself that it possesses any real advantages. The fact is, that mercury in any form is so prompt in its results against syphilitic inflammations, that whoever is in the habit of prescribing it in one special form is very apt to come to the conclusion that his peculiar mode of use must be superior to others.

When there is a rash or ulceration on the cutaneous surface, it is always well to apply mercury directly to the inflamed part in addition to its internal use. This may be done conveniently by prescribing the mercurial ointment when there is no ulceration, and the black wash when the latter exists. Of the various preparations of mercury, I much prefer calomel or the bichloride, and rarely find that these when used with suitable adjuvants, in moderate dose, disagree to any material amount. For the early forms of secondary rash I usually employ mercury alone, and for the latter ones mercury in combination with iodide of potassium. A mixture containing the solution of the bichloride with the iodide in excess is extremely useful. It probably amounts to bin-iodide of mercury with excess of iodide of potassium. The addition of ammonia to this mixture appears to increase its efficacy, and the same is the fact as regards its use with the iodide of potassium under all circumstances.

In prescribing mercury it is the surgeon's duty to carefully watch its effects upon the disease, and upon the patient's health. The fact that a given patient is cachectic and feeble is not in itself any reason for precluding resort to specific treatment. On the contrary, it is under such circumstances not infrequently that the value of the remedy is best shown. Nor can we lay down any rule as to the avoidance of mercury in treating certain forms of rash,—ecthyma, rupia, &c. It may be stated in general terms that the more nearly the rash keeps to the scaly type, the more certain is it that mercury will agree; whilst the greater the tendency to ulceration and suppuration, the more is the risk that it may disagree. Should it disagree in any case, the observant surgeon will soon discover the fact. If the ulcers spread instead of healing, and if the quantity of secretion is increased, then mercury should be at once laid aside and substituted by iodide of potassium with tonics. The cases in which this substitution becomes necessary are, I believe, often those in which the patient has inherited partial immunity, and consequently suffers from a modified form of the acquired disease. Although mercury is very efficacious in the infantile stage of Congenital Syphilis, it often disagrees most markedly with the subjects of this taint when they have attained adult age. As a rule, in the management of Secondary Syphilis, it is well to push mercury to a mild degree of ptyalism. Not infrequently symptoms resist its action until the gums are sore, and then yield at once. I have often observed this in inflammation of the eye, especially in retinitis.

With regard to the iodide of potassium against tertiary symptoms, a few simple rules may be given. It is well always to combine it with ammonia. It is well to begin with a small dose, *e.g.* five grains three times a day, and to gradually increase, not going beyond ten or twelve unless necessary. If the disease does not yield to the latter, the diagnosis being yet undoubted, then much larger doses should be given; say, half a drachm three times daily. In a few rare cases nothing short of these large doses will produce any benefit.

THE DIAGNOSIS OF CONSTITUTIONAL SYPHILIS.—The power of recognising syphilitic diseases when brought under notice is one of the most valuable gifts which the physician can possess. These diseases meet us at every turn in practice, and present a most bewildering variety of external aspect. At one time we have to distinguish a syphilitic rash from a simple one, at another to diagnose between Syphilis and Cancer, or Syphilis and Rheumatism, or the problem presented may be to form a correct opinion as to the nature of a paralysis, an ascites, or an attack of mania. The first requisite to success is a mind constantly awake to suspicion, and fully impressed with the all-important fact that diseases of the most diverse character may have their origin in this taint, and that if so they will prove to be curable only by treatment directed against it. This suspicion must be present, whatever may be the position in life or the reputation of our patient. A gentleman, who now at middle age bears the most irreproachable character, may chance to have been less circumspect during college life, and it is not fair that his subsequent purity of conduct should be the means of preventing his relief from the consequences of youthful error. We meet every day with cases in which women, whose characters are spotless, have become the subjects of syphilitic taint without their having the slightest suspicion as to the nature of their malady. Under many circumstances it is out of the question to make any direct inquiries, and the physician must depend upon his own acumen for the opinion which shall guide his treatment.

The recognition of syphilitic symptoms in the *secondary stage* is not usually difficult. The copiousness of the rash; its symmetry; the copper-tint; the frequent coincidence of several of different types of skin eruption in the same case; the presence of febrile disturbance; the absence of cutaneous irritation and the co-existence of sores on the tonsils, and frequently on the mucous membrane of the cheeks also—are all features which help to make the diagnosis easy and certain. To these we may add that the syphilitic exanthem usually appears first on the abdomen, chest, and fronts of the arms, that it very commonly affects the face, and that it avoids the backs of the elbows and the fronts of the knees, localities which are almost always attacked in cases of common psoriasis. Although syphilitic rashes vary very much in outward characters, yet they have always in the features just mentioned a basis of close similarity. When mistakes occur they are usually those of insufficient attention. The patient is allowed to show only a small part of his surface, instead of being made to strip, or at any rate to expose the whole of his bust. If the latter course be adopted, the symmetry of the rash and its other peculiar features will almost always arrest the attention of the observer. Amongst minor points which occasionally assist may be mentioned the gyrate or ringed form of the patches, and in some cases of syphilitic psoriasis the comparative absence of desquamation.

The cases which cause most difficulty are those in which Syphilis occurs in a patient who is already the subject of some other skin disease. In hospital practice it is very common to see scabies and a syphilitic rash co-existing, and in some such it is most difficult to pronounce with certainty as to the nature of the eruption. Examination of the mouth and throat and of the genitals will often remove doubt; but if not, a few sulphur baths as a measure of diagnosis will usually prove successful.

Next to that of the exanthem itself comes the diagnosis of the *relapses of eruption*, which often occur between the secondary and the tertiary epoch. In these there is rarely any copious outbreak, usually only a few isoalted patches. These are most commonly met with in the palms of the hands or soles of the feet, or on the front aspects of the forearms or legs. They are almost dry and attended with peeling of the epidermis. Very frequently there are small sores in the mouth or on the tongue; at the same time a form of acne, chiefly affecting the forehead, and leaving little pits or scars, is very often seen in this stage. If iritic adhesions are present, or if there are pits in the skin of the face and trunk, left by a former rash, the suspicion is much strengthened. If the diagnosis remains doubtful, we may cautiously try the effect of a short treatment with the bichloride or biniodide, and observe the result.

Lastly, we must consider the question of the recognition of the various diseases which come into the category of *tertiary symptoms.* When these occur, it is often many years since the patient has suffered from any other, and it is quite possible that he may appear to be in excellent health. A few of the tertiary symptoms have been so long recognised in relation to their true cause, and are so rarely met with in connexion with any other, that in themselves they almost constitute their own diagnosis, and often also help us to that of more obscure lesions. Periosteal swellings or nodes are the chief of these. To speak generally respecting other forms we may say that the diagnosis must be founded in part upon the patient's previous history, in part upon any still existing remnants of former disease—such, for instance, as iritic adhesions—and in part upon the peculiarities of the disease itself. As regards the patient's antecedents, I may just remark, by way of caution, that we must not hastily assume that he is syphilitic because he tells us that he has had the venereal disease. A soft chancre with its suppurating bubo, or even an attack of gonorrhœa, although both of them quite innocent as regards constitutional infection, often leave more vivid impressions on the patient's mind than does an indurated sore and its exanthem. Those who are most ready to suspect a venereal cause, are often those who have never had true Syphilis at all. If, however, there is a clear history of a chancre followed by secondary rash, sore throat, &c., then we have obtained a fact which, whatever may be the present ailment, may be safely permitted to modify our treatment. The majority of tertiary lesions are by conventional usage regarded rather as surgical than medical, and it would be out of place to speak in detail of the diagnosis of ulcers, gummous tumours, &c. I may briefly remark that the serpiginous form of ulceration, healing in the centre and spreading at the margin, is a feature always to be regarded with suspicion; that tumours in muscle, which will wholly melt away under the influence of the iodide, are sometimes as hard and as defined as any variety of cancer, and have often led to needless operations.

In cases of disease of the nervous system in which Syphilis is suspected, an examination of the patient's eyes, throat, tongue, and tibiæ should never be omitted. The existence of iritic adhesions, of cicatrices of the soft palate or of periosteal nodes, will often decide the question. The occurrence of nocturnal exacerbations of pain is also always suspicious. If the disease implicate only one nerve-trunk, especially if only one cranial nerve be involved, the suspicion of Syphilis becomes very strong. Probably a full half of the cases of paralysis of the third, fourth, fifth, and sixth nerves, when such paralysis affects only one nerve, are due to Syphilis, and are curable by specific treatment. In these cases the disease is hardly ever symmetrical, and the paralysis is usually complete. The seventh nerve is occasionally attacked, but not so frequently as the others. The nerves of special sense are not so frequently affected in acquired Syphilis as they are in the inherited form. Nevertheless, cases do occasionally occur in which amaurosis or complete deafness are met with in the subjects of syphilitic taint, and without other assignable cause. In these the loss of function is usually symmetrical, and probably depends upon disease of the cerebral centre rather than neuromata developed in the nerve-trunks. I am not aware of any cases in which paralysis of the branches of the eighth pair have been traced to Syphilis, but no doubt such occur and might be recognised by due search. Paralysis of single nerve-trunks of any of the spinal plexuses —more especially of those of the brachial plexus—are now and then encountered.

In cases of Tertiary Syphilis the bones of the calvaria not infrequently increase greatly in thickness and weight without any formation of external node, and under such circumstances there is very often a roughened state of the surface in contact with the dura mater. Various forms of disturbance of the sensorial functions are usually observed in these cases. In addition to violent headache there is irritability of manner, loss of memory, and sometimes actual mania.[1] The diagnosis must depend upon the facts to which I have already adverted.

THE DIAGNOSIS OF CONSTITUTIONAL SYPHILIS WHEN CONSEQUENT UPON INHERITED TAINT.

The diagnosis of inherited Syphilis rests on somewhat different data to that of the acquired disease. Indeed, the whole course of the disease, as thus transmitted, presents some remarkable features of difference which I have endeavoured to bring into clear contrast in the appended tabular parallel. (See p. 761.) Some local lesions, not infrequent in those who have inherited the taint, never occur at all in those who have acquired it, as for instance, interstitial keratitis. Others present important modifications of character; thus, when periosteal nodes occur in children, they are much more extensive than is usually the case in adults. Speaking generally, the tertiary symptoms of inherited Syphilis, however long may have been the interval of latency, are for the most part symmetrical. We have seen that those of acquired Syphilis are but rarely so.

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[1] A young man was recently admitted under my care into the London Hospital, having attempted suicide (by cutting his throat) in a state of mania. He had disease of the alveolus and nasal bones, which led me to diagnose Syphilis. We gave him full doses of iodide of potassium, and he rapidly recovered. When his mental faculties had returned, he gave us a history which fully confirmed our suspicions as to specific taint.

The stages observed in the course of inherited disease are very similar to those of the acquired form, but they much more frequently run into each other.

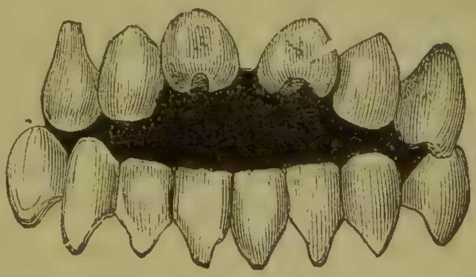
We must consider the question of diagnosis in reference to the three different stages: first, the infantile period; second, the stage of latency; and lastly, that of tertiary symptoms (usually about the age of puberty).

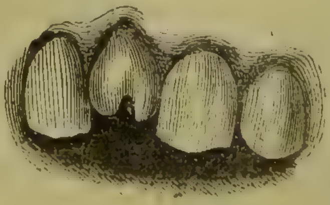
In the infantile period we recognise Syphilis by the peculiarity of certain single symptoms, or, more frequently, and with greater certainty, by the peculiar grouping of several different symptoms. First in importance is the rash on the skin. The rash, as in acquired Syphilis, may vary much in its character, but the commonest are those of erythematous or papular character. If it is erythema, the redness will show itself in abruptly margined patches, and will be characterised further by its peculiar red or coppery tint, compared by some authors to that of the lean of ham. Sometimes we see instances of dry, scaly rashes in infants, but these are rare. Pustular, vesicular and bullous rashes are also not infrequently witnessed. Condylomata at the anal orifice are common, though less frequent during the first few months than at later periods. At the same time as the rash the little patient almost always displays the characteristic symptom known as "snuffles," and there is usually inflammation of the mucous membrane of the mouth, and sores at its angles. (See p. 740.) Iritis occurs in a few cases and has similar tendencies to those witnessed in the acquired form, and is equally under the influence of specific treatment. It occurs also at the same stage, always amongst the secondary symptoms. Inflammations of the deep-seated structures of the eye—of the vitreous, retina, choroid—are as frequent as they are in the adult, and present the same characters.

During the stage of outbreak of the exanthem, which lasts on the average from the fourth week to the sixth month, the child becomes fretful, pale, and emaciated; growth is for a time arrested, and his shrivelled face resembles that of an old man. Emaciation is certainly the rule, but it has many marked exceptions, and I have often seen syphilitic infants who were fat and plump and looked remarkably well.

At or about the age of one year, if the child have survived, it is usual for the secondary symptoms to wholly disappear. The period of latency now ensues, during which the child enjoys often very good health. Sometimes relapses occur, and especially are such subjects liable to be affected by condylomata. These relapses scarcely ever involve a return of cutaneous rash. I think that all observers will bear me out in the statement that the characteristic rashes so often seen in syphilitic infants are never witnessed at later periods of life. The tertiary epoch may begin at any period after the fifth year, but it is commonly delayed till at or near the period of puberty.

The recognition of the subject of inherited Syphilis, at or after the age of puberty, may be sometimes made with great certainty, and is at others surrounded by difficulties. Our most valuable aids are the evidences of past disease, more especially of the inflammations which may have occurred in infancy. A sunken bridge of nose, caused by the long-continued swelling of the nasal mucous membrane when the bones were soft, a skin marked by little pits and linear scars, especially near the angles of the mouth, the relics of an ulcerating eruption, and a protuberant forehead, consequent upon infantile arachnitis, are amongst the points which go to make up what we recognise as an heredito-syphilitic physiognomy. Added to them we have very valuable aid furnished by the shape of the incisor teeth. In these patients it is very common to find all the incisor teeth dwarfed and malformed. Sometimes the canines are affected also. These teeth are narrow and rounded and peg-like; their edges are jagged and notched. Owing to their smallness their sides do not touch, and interspaces are left. It is, however, the upper central incisors which are the most reliable for purposes of diagnosis. When the other teeth are affected these very rarely escape, and very often they are malformed when all the others are of fairly good shape. The characteristic malformation of the upper central incisors consists in a dwarfing of the tooth, which is usually both narrow and short, and in the atrophy of its middle lobe. This atrophy leaves a single broad notch (vertical) in the edge of the tooth, and sometimes from this notch a shallow furrow passes upwards on both anterior and posterior surface nearly to the gum. This notching is usually symmetrical. It may vary much in degree in different cases; sometimes the teeth diverge and at others they slant towards each other. The appended woodcut illustrates a good example of the deformity. In any case in which the malformation was as marked as in this sketch, I should feel no hesitation in pronouncing the possessor of the teeth to be the subject of inherited Syphilis even in the absence of other testimony. I have never yet seen such teeth, excepting in patients of this class. In the majority of cases, however, the condition of the teeth is sufficient only to excite suspicion and not to decide the question. In a few rare cases only one of the upper central incisors is malformed, the other being of natural shape and size. A good instance of this state of things is shown in this woodcut.





In a considerable number of cases of heredito-syphilis the teeth show no deviation whatever from the normal standard, and in such the diagnosis must be guided by other conditions. In addition to the peculiar malformations above described and illustrated, there are others which, although less characteristic, are yet very valuable to a trained observer. They do not, however, admit of description without great risk of misleading the reader. Before leaving the subject of dental malformations, I may again ask attention to the fact, that it is only in the permanent set that any peculiarities are observed. The first set are liable to premature decay, but are not malformed.

In addition to the peculiarities of physiognomy and the malformations of the teeth, the diagnosis may be much helped by observing the state of the eyes and of the bones. If there be evidences of past iritis, or if there be clouds in the substance of the corneæ, the results of past keratitis, or especially if the corneæ be now attacked by this peculiar inflammation in its acute stage, very valuable evidence will have been obtained. The phenomena of syphilitic keratitis in its acute stage are peculiar and easily recognised. Both eyes are usually affected at the same time. The corneal tissue becomes very extensively opaque by the effusion of lymph into its substance. Its tint may vary from that of ground glass to a red salmon colour. There are no ulcers on its surface. A zone of ciliary congestion is usually well marked. The patient is often for several months, whilst the disease is at its height, practically blind. The intolerance of light is usually considerable. After the inflammation has passed away the cornea usually clears in a most remarkable manner, but it rarely regains such perfect transparency that the experienced observer cannot detect traces of what has taken place. These traces consist in a somewhat dusky and thin sclerotic in the ciliary region, and in the presence of slight clouds here and there in the corneal substance, there being no scars in its surface. The difference between these interstitial clouds and ordinary leucomata is easily observed.

In a few cases the existence of nodes on various long bones may help us to a diagnosis, and in others we may obtain aid from finding that the patient has become deaf without otorrhœa, or that he is partially amaurotic from choroiditis.

With regard to the general arrest of development in heredito-syphilis, I may remark that it is a very untrustworthy indication. In a few cases this taint dwarfs the whole body in a most remarkable manner, but in most cases no retardation of general growth is observable. A pale complexion is almost always met with. It is exceedingly rare to meet with a florid good complexion, in a young adult who is the subject of this taint. We do, however, every now and then see a physiognomy which neither in shape of features nor in colour of cheeks and lips furnishes the slightest clue. I have met in one or two instances with arrest of sexual development. In one of these, a young woman under the care of Dr. Hughlings Jackson in the London Hospital, there was such an entire absence of all sexual characteristics, that I could not but suspect that the ovaries had been destroyed by syphilitic inflammation in early life.

CONTRASTED PARALLEL BETWEEN THE COURSE OF SYMPTOMS IN ACQUIRED AND INHERITED SYPHILIS.

I have endeavoured in the following tabular statement to place as clearly as I can the resemblances and differences which we observe in the course of symptoms when arising from acquired or from inherited taint. To some of these I have already incidentally alluded, and respecting the others the statements in the Table will, I trust, explain themselves:

ACQUIRED DISEASE.

Primary Stage.—Local or tage of inoculation. The sore appears after an incubation period of from ten to twenty-eight days, and if not treated may remain from a fortnight to six months. Liable to relapse.	An ulcer (chancre) usually with indurated base. Indurated lymphatic glands. Induration is to be regarded as the earliest proof of successful inoculation, but the latter is sometimes effected without any hardness of the original sore having shown itself.
Secondary Stage.—Constitutional or exanthematic. Usually commences within six weeks or two months of the inoculation, and if not treated, may last from three to six months or to a year. Essentially transitory, and will disappear without treatment.	Febrile disturbance, malaise and muscular pains. Slight engorgement of lymphatic glands in many parts. A symmetrical, and usually copious eruption on the skin, and often on exposed mucous surfaces. Symmetrical ulcers in tonsils. Iritis, retinitis, &c., usually symmetrical. Loss of hair, loss of flesh and of strength. This stage may be either exceedingly slight or very severe. Its severity appears to bear proportion to the degree of induration of the preceding chancre. It is often noticed that the rash comes out in successive crops. The rash may also vary very widely as to its character, roseolous, scaly, papular, pustular, ecthymatous, &c., being modified probably by peculiarity—first, in the source of contagion; secondly, in the state of health of the recipient.
Intermediate Stage.—Stage of latency and of relapses. This stage may be said to commence at from a year to a year and a half after the contagion, and to extend over a period which may vary from three to five, ten, or even twenty years.	The patient may be either wholly free from symptoms and in good health, or he may remain pale and rather feeble, and liable from time to time to slight returns of eruption on the skin, sores on the mucous membranes, condylomata, &c. He is protected as regards fresh contagion, and should he beget children they are almost certain to suffer. The relapses during this stage are usually

easy to be distinguished from true secondary symptoms. There is little or no febrile disturbance, the rash is not copious, and often not symmetrical. Acute iritis, retinitis, &c., never occur; that is, they do not occur for the first time; they may occur in the form of relapses.

Tertiary Stage or stage of sequelæ.

This stage commences at from four to ten or to twenty years after the contagion, and extends indefinitely, very often to the end of life.

All the symptoms in this stage occur, as a rule, without symmetry; sometimes multiple, but not infrequently single. They consist of chronic inflammations of deep tissues, or of the deeper layers of superficial ones, *e.g.*:— Inflammations of periosteum and bone resulting in nodes; of cellular tissue, tendon, or muscle, resulting in gummy tumours; ulcerative destruction of the palate and pharynx; serpiginous ulcerations of the skin; inflammations of nerves, or even of cerebro-spinal centres, inducing various forms of paralysis; deposits in liver, lungs, &c. Probably but little liability to transmit the disease to offspring. Protection against a new contagion incomplete. All the inflammations in this stage are remarkably under the influence of treatment by iodide of potassium, but tend to relapse. Unless so treated, all of them tend to progression and permanent disorganization of the part attacked, none of them to spontaneous recovery.

INHERITED DISEASE.

Primary Stage.

The infants usually remain without symptoms for from one week to three months.

This stage has been passed through by one or both of the sufferer's parents within from a few months to twenty years of the infant's birth. The infant is usually free from all symptoms at the time of birth.

Secondary Stage.— Constitutional or exanthematic.

From the age of two to four weeks to the end of the first year.

This stage is essentially transitory, and will disappear without treatment, if the child lives.

Inflammation of nasal mucous membrane causing "snuffles."

A symmetrical and usually copious eruption on the skin. Wasting; fretfulness; a peculiar odour; a withered, senile aspect; inflammation of the mouth, and condylomata at anus; iritis, usually symmetrical; arachnitis and slight effusion; disease of liver (rare); nodes (very rare). The eruptions which occur differ from those of acquired disease, chiefly in being more moist, and in preferring the thighs and genitals. These differences may in part be due to peculiarities in the skin of young infants, and to the constant irritation from urine to which the nates are liable. Dry scaly rashes are rare. Iritis is much less frequent than in the adult, but just as well characterised when it does occur.

In infants this stage often proves fatal.

Intermediate Stage.— Stage of latency.

This stage extends from the end of the first year or eighteen months to the second dentition, the time of puberty, or even very much later.

The patient will probably be wholly free from active symptoms, but will show various indications of his diathesis in pallor of skin, sunken nose, protuberant forehead and premature loss of the upper incisor teeth. Sometimes there will be a remarkable retardation of growth and general development. If second dentition have occurred, the central upper incisors will be malformed. Unlike what happens during this stage in acquired syphilis, we scarcely ever observe any tendency to recurrence of the secondary symptoms. Now and then we see condylomata at the anus returning during the first five years, but the rash of infantile syphilis having once disappeared, I think scarcely ever relapses. A certain degree of nasal obstruction sometimes persists, but not often.

Tertiary Stage or stage of sequelæ.

This stage may commence with the second dentition, at the time of puberty, or not till much later. Its duration is quite indefinite.

Most of its symptoms are symmetrical:—

Keratitis (interstitial); kerato-iritis; periosteal nodes; cerebral deafness (not infrequent); cerebral blindness (rare); disease of liver and kidneys; phagedænic or serpiginous ulcerations of skin; cellular nodes (rare). Probably not liable to transmit the disease to offspring. Protection against a new contagion incomplete. The symmetry of the symptoms is in marked contrast with what occurs in this stage of acquired disease. The paralyses of single cranial or spinal nerves, so common from acquired syphilis, are, I believe, never met with in the inherited form.

Most of the inflammations tend, unless arrested by treatment, to permanent disorganization, but one (interstitial keratitis) tends to recovery even without treatment. They are much less easily influenced by treatment than those of the acquired disease.

GENERAL DISEASES, OR AFFECTIONS OF THE WHOLE SYSTEM, *continued.*

§ II.—*Those determined by conditions existing within the body:—*

SCORBUTUS.
PURPURA.
RICKETS.
GOUT.
RHEUMATOID ARTHRITIS.
RHEUMATISM.
GONORRHŒAL RHEUMATISM.

SCORBUTUS.

By Thomas Buzzard, M.D.

Scurvy, or Scorbutus, as it is technically called, is a peculiar state of mal-nutrition, supervening gradually upon the continued use of a dietary deficient in fresh vegetable material, and tending to death, after a longer or shorter interval, if the circumstances under which it arose remain unaltered. The condition is essentially marked by a dull leaden pallor of complexion; excessive bodily debility and mental lethargy; dyspnœa upon slight exertion, unaccounted for by the auscultatory signs; spontaneous effusions of blood-coloured fluid into the various tissues of the body, causing petechiæ and bruise-like patches to appear on its surface; together with (commonly) a livid, swollen, and spongy state of the gums, and a disposition for them to bleed upon the slightest irritation.

Synonyms.—*French*, Scorbut; *German*, Scharbock; *Italian*, Scorbuto; *Spanish*, Escorbuto. These terms, as well as the English Scurvy, take their origin from the Danish Skörbeck, "Disease of the mouth," of which the word "Scorbutus" is a barbarous Latinized version.

Etiology.—Very much has been written upon the subject of Scurvy. Long before the disease was styled by the name which it now bears, and when, indeed, it was often called the "unknown disorder," historians noted its ravages in armies located, under circumstances of difficulty, in foreign lands, or in garrisons shut off from obtaining supplies of requisite nutriment. In the long sea-voyages which the intrepid navigators of the fifteenth and sixteenth centuries commenced, the crews suffered terribly from a disorder which destroyed the lives of large numbers, and affected the working power of the survivors in a manner equalled by no other disease. It is probable, indeed, from the records which have come down to us, that Scurvy, either alone or as influencing the severity of accompanying maladies, has proved more destructive to mankind than any other disorder. There is no more interesting fact in the history of medicine than that this condition, which has been looked upon at various times as plague, as a mysterious infliction of Divine justice against which man could only strive in vain, or as a disease inseparable from long voyages, should have been proved, by evidence of the most satisfactory character, to arise from causes in the power of man to prevent, and to be curable by means which every habitable country affords.

Scurvy only occurs when fresh vegetable nutriment has been for some time partially or completely withheld. A variety of forms of impaired nutrition will follow the want of other descriptions of food, but this particular condition is only seen as a sequel of that special privation. Scurvy does not occur when the supply of wholesome and fresh vegetables is abundant, even though the food generally may not be adequate to perfect nutrition. There is a degree of positiveness about these two assertions which can rarely be ventured upon in the etiology of disease. In this case, so abundant and conclusive are the proofs, that to assert less strongly would be to imply a doubt which cannot be allowed to exist. The grounds for these statements are to be found in the voluminous records of the circumstances under which the disease has occurred, as related by observers, either unbiassed by any theory of their own, or widely differing in their opinions as to the cause of the disorder. They may be said to form the negative side of the argument. The affirmative is based upon the fact, universally allowed by those largely experienced in the disease, of the power possessed by fresh vegetable material, and by that alone, in removing the disease.

It is not our intention to quote at length the records of Scurvy outbreaks, in proof of these assertions. We shall but glance at some of the more prominent instances in recent times, referring the reader for more elaborate details to the vast bibliography published upon the subject by writers of almost every nation. In the exhaustive treatise upon this disease, by Dr. Budd, in the Library of Medicine, will be found a history of Scurvy, which has served more than any other publication to place the disease in its true light, and the views expressed in it have been remarkably confirmed by the experience of the several outbreaks which have occurred since it was written.

In 1846 the potato crop failed in the United Kingdom. In the autumn, winter, and following spring, numerous cases of Scurvy occurred amongst all classes of society. Dr. Christison has described[1] an outbreak of the disease amongst the labourers employed upon the Scotch railways. Their food consisted of bread, salt pork, butter, cheese, coffee, tea, and sugar. "Potatoes were, of course, out of the question. Fresh vegetables were never thought of, and were, indeed, in most places inaccessible." The quantity of food seems to have been sufficient. In the Royal Infirmary of Glasgow, 83 cases were admitted. Dr. Ritchie[2] writes: "The general fact in regard of the food of all was that it failed in variety, and in the quantity of its animal constituents, and that in all but a fraction of the cases in which they were very deficient, the patients had been exposed for months to a total deprivation of fresh succulent vegetables." "In Carlisle and its vicinity," Dr. Lonsdale informs us,[1] "the persons chiefly afflicted were weavers and their wives and daughters working in the factories, shoemakers, and comparatively few of any other kind of artisans. Bread, oatmeal, treacle in very small quantities, tea and coffee, with an occasional herring, formed their entire food. None had tasted potatoes after the harvest of 1846, or for a period of seven or more months." Dr. Lonsdale states also that in a great number of the huts occupied by the railway excavators, amongst whom there were numerous cases of the disease, some of which proved fatal, he saw the men breakfasting off beef-steaks or mutton-chops and bread. The dinner comprised bread, boiled beef or bacon, pea soup or broth, and suet puddings containing currants. The animal food was taken in large quantities; there were no potatoes or fresh vegetables. At Workington (a seaport town of seven thousand inhabitants) the disease did not show itself. Dr. Dickinson, a resident, assigned as a reason, "that vegetable food was more abundant there than in many situations, particularly turnips, of which large quantities were used."

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[1] Edin. Monthly Journal, July 1847.

[2] Ibid.

The same kind of evidence is adduced by Dr. Curran[2] in his description of the occurrence of the disease in Ireland. "In four-fifths of the cases reported to me, bread and tea or coffee was what the patients had been living on when attacked; the others had been using grains of various kinds, or grains and flesh or fish; but *in no single instance* could I discover that green vegetables or potatoes had formed a part of their regular dietary." Dr. Shapter[3] remarks, in reference to the cases of Scorbutus observed by him in Exeter, that the only difference from the usual diet of the sufferers consisted in the absence of the potato.

There is no doubt that the failure in the potato crop, besides depriving the population of this vegetable, incidentally also rendered their nutrition imperfect, by increasing the price of provisions in general. This circumstance might naturally, therefore, be believed to bear its part in the causation of this particular morbid condition, were it not that the disease was not confined to the poorer classes of society. Dr. Shapter relates that many of his patients were persons who had experienced no difficulty in procuring an abundance of the necessaries of life, with the exception, however, of potatoes or fresh vegetables. Dr. Christison notes the occurrence of Scurvy amongst railway labourers "earning ample wages, and whose extravagance in good living was a frequent subject of remark in their neighbourhood."

The allied armies of England, France, Turkey, and Sardinia suffered severely from Scurvy in the Crimea and Asia Minor during the war with Russia, 1854-56. The disease first began to show itself amongst the British troops in Bulgaria, when they had been living for some months upon an inferior diet, with but a very scanty supply of vegetables. On their arrival in the Crimea the men found an abundance of grapes, cabbages, &c., which were eagerly consumed; and although the rations in other respects were most imperfect, no cases of the disease were recorded in September. As the winter advanced, however, and the vegetable food ceased, the affection reappeared. It was no part of the ordinary duty of the Commissariat to supply vegetables, which did not constitute a part of the soldier's rations. In consequence of representations, a supply of lime-juice was ordered, and arrived by the *Esk* on the 10th December. By one of those accidents, of which so many lamentable instances occurred during the early part of the war, no portion of the juice was issued until the first week in February 1855, and then the supply was very insufficient. In March nearly all the sick arriving at Scutari from the Crimea were suffering from Scurvy. The total admissions from Scurvy during the war amounted to 2,096, but "the returns convey but a faint conception of the disastrous part which it acted among the troops, for although it comparatively rarely presented itself in well-defined forms, and as an independent affection, yet the prevalence of scorbutic taint was wide-spread, and in a vast proportion of cases evident indications of it existed as a complication of other diseases, especially fever and affections of the bowels."[1] As the supply of fresh vegetables and lime-juice became more constant, the disease gradually disappeared, and comparatively few cases occurred during the second winter.

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[1] Edin. Monthly Journal, August 1847.

[2] Dublin Quarterly Journal, 1847.

[3] Lond. Med. Gazette, vol. iv.

The sufferings of the French from this cause were proportionally much greater than those of our troops. This must be ascribed to the fact that the distribution of lime-juice formed no part of their practice. In fact, even at the present time, the French authorities do not appreciate at its full the value of this addition to the diet of either soldiers or sailors. No less than 23,000 cases of Scurvy are recorded as occurring amongst the French troops.[2] From the month of February 1855, fresh meat was supplied to them at first twice and then five times a week. It was of good quality, but lean. The supply of bread was irregular, and fresh vegetables formed no part of their rations. Rice was allowed, and occasionally dried vegetables, principally peas, beans, and lentils, figured in the diet, but in small proportions.[3] There was this which was remarkable in the outbreak:—The disease first showed itself in the winter of 1854-55, and committed terrible ravages. As the season opened and the earth began to bring forth vegetation, Scurvy diminished. The troops were encouraged to collect herbs, and especially dandelion, which was very plentiful, and of which the effects are highly anti-scorbutic. The improvement which took place under these circumstances was very marked, and there is no reason to believe that it would not have been permanent had the supplies continued. But as July approached, the rays of the sun dried up the surface of the ground on which the troops were encamped, no more dandelion was to be obtained, and in the course of three months, the finest and warmest in the year, no less than 5,000 cases of Scurvy occurred. It may be remarked here, incidentally, that this outbreak furnishes a striking contradiction to the theory which ascribes the scorbutic condition to the influence of cold and damp.

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[1] Med. and Surg. Hist. of the British Army, 1858. By Authority.

[2] Rélation Médico-Chirurgicale de la Campagne d'Orient, par le Dr. G. Scrive. Paris, 1857.

[3] L'Union Médicale, 1857, p. 419: M. Perrin.

The Sardinian army, which arrived in the Crimea in the early summer of 1855, was very generally affected by Scurvy. Its ravages were checked by vegetables supplied to the troops as the season advanced.

It is most probable that the Turks experienced even more severe losses from Scurvy than our other allies; but, from the imperfect organization of their medical department, exact statistics are wanting. There is no doubt, however, that the original force which formed part of the expedition from Bulgaria to the Crimea, was almost entirely swept off by disease, of which Scurvy formed an important element. Of the fresh troops, under the command of Omer Pasha, which reinforced these men, and which were posted at Eupatoria during the winter of 1854–5, as many as 1,000 were sent down monthly to Varna, all of whom were suffering severely from this cause, and a very large number of whom died upon the passage. During the summer the remainder, amounting to about 20,000, were encamped in the neighbourhood of Balaklava. We had ourselves the opportunity of observing them narrowly. Their food was very imperfect; but they showed ingenuity in availing themselves of such fruits and vegetables as were obtainable, and they were supplied with onions. They consumed, especially, large quantities of water-melons, which were procured from the Tartars inhabiting the country, or from sutlers. No cases of Scurvy fell under our observation at that time, nor could we detect any scorbutic taint in patients suffering from other diseases. In November they quitted the Crimea, and campaigned in Mingrelia. During the winter all supplies of vegetables ceased, and, with the early spring, shiploads of sick were brought to Trebizond, all of whom were severely affected with this disease. Large numbers died upon the sea-passage, in their transit from the shore to the hospital, and soon after their admission. The sick included numerous examples of Scurvy, developed to an extent which recalled the terrible descriptions of the disorder contained in the narratives of our early voyagers. Such cases as these were rare amongst the other allied troops. In explanation of this fact, it must be noticed that throughout the winter fresh vegetable food had formed no part of the rations distributed to the Turks, and, superadded to this, was absolute starvation, from the absence of food in sufficient quantity. Their diet, indeed, had consisted entirely of biscuit, a little rice, haricot beans, and "yagh," a coarse butter made from mutton fat.

Dr. Hammond informs us that, during the recent war in America, no confirmed Scurvy appeared amongst the Federal forces, but a scorbutic taint often manifested itself. He attributed its occurrence to occasional deficiency in the supply of vegetable food, exposure to cold and damp, and mental depression. He did not find that salt meat had any influence in its production. If the men had vegetables they could eat salt meat with impunity. Raw potatoes preserved in molasses were commonly issued to the troops, and were found of signal service in preventing the disorder.

So also, on the Confederate side, Dr. Darby, late medical director, in a communication which he has been kind enough to send to us, says, "The type of the disease characterised by petechiæ and spongy gums was rarely known in the Confederate army. Diseases and injuries incidental to army life assumed at times a scorbutic taint, at such seasons and under such conditions as give rise to this malady. The abundance of proper supplies in the early stages of the war prevented scorbutic tendencies. In proportion as a decrease of supplies took place, there was an increase of the scorbutic type in all disease."

Since the year 1795, Scurvy, unless under very exceptional circumstances, has been all but abolished from the British fleet, and for this the name of Dr. James Lind, "the father of nautical medicine," deserves to be held in lasting honour. The combined observation of exploring navigators had tended to show that the disease could be cured by supplies of fresh vegetables, and its occurrence prevented by a similar diet. It was reserved for Dr. Lind, in his celebrated work on Scurvy published in 1753, to give overwhelming proofs of the efficacy of lime-juice as a prophylactic in this disease, and forty-two years afterwards (!) the Admiralty took the hint. To Captain Cook, especially, science is indebted for a practical exposition of the influence of vegetable food. By providing his crew with abundance of sauer-kraut, and encouraging them to seek wild vegetables wherever he landed, he preserved their health completely during a four years' voyage of his ship *Discovery*. The same principle, though in the more convenient form of a daily ration of lime-juice, suffices to prevent the disease in the royal navy. The merchant service still, however, continues to furnish cases of this preventible disorder. Although the Legislature insists, under a penalty, that lime-juice shall be issued to the crews, the provisions of the Act are but too frequently evaded, and the Dreadnought Hospital still continues to receive annually an average of ninety cases of the disease—about one in twenty-five of all patients admitted.[1] Practically, in many cases, no lime-juice is furnished; or a cheap imitation of the juice, consisting of tartaric acid, sugar, and water, flavoured with essence of lemon, is substituted. On the other hand, emigrant and convict vessels sailing to Australia, being under more complete Government supervision, convey their passengers without loss from the disease.

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[1] There is an excellent article upon this subject by Dr. Barnes in the Sixth Report of the Medical Officer of the Privy Council, 1863. See also an able report by Mr. Harry Leach, resident medical officer to the *Dreadnought* in a House of Commons' return, "Scurvy in Merchant Ships," June 1865. Mr. Leach ascertained by inquiry that of eighty-three Scurvy patients admitted in 1864, forty-two had received bad lime-juice, eighteen had had none at all; fourteen could give no exact account; and in the case of nine only was good lime-juice declared to have been taken.

Now, it may be argued against such facts as we have recorded, that inasmuch as these outbreaks of Scurvy have always occurred amongst persons in an unnatural state of existence, in periods of famine, landed with insufficient provision in an enemy's country, or cooped up on board ship in long voyages, there may be some other special privation to which the occurrence of Scurvy is quite as likely to be due. This argument, in fact, has been repeatedly employed, and even at the present time it is still occasionally urged. In opposition to the view which we advocate, several causes of Scurvy have been insisted upon by various writers. Some have attributed the disease to the action of some one noxious agent, whilst others, and this is more common, urge that a combination of circumstances is necessary for its production. The point upon which there still exists the most important difference of opinion is regarding the influence of *salt meat.* There is no doubt that, in the great majority of Scurvy outbreaks, salt meat has formed an important part of the food taken by the sufferers. But, in order that this circumstance should be possessed of any weight, it would be necessary to show that the disease was never known to occur unless this description of food had formed at least a part of the dietary. There is evidence in plenty to the contrary.

During the campaign of Louis IX. of France, in Egypt, 1249, the army was frightfully ravaged by Scurvy, of which a most graphic description is afforded us by the historian.[1] During Lent, which was very strictly observed, the troops ate no meat, but subsisted "on eel-pouts, which is a gluttonous fish."

During the war in Hungary, in 1720, between the Austrians and Turks, many thousands of the former were cut off by Scurvy. Kramer informs us that they ate no salt beef or pork; but, on the contrary, had plenty of fresh meat at a very low price.[2]

The French prisoners confined in Sisinghurst Castle, in Kent, in the middle of the last century, suffered much from Scurvy. They had eaten no salt provisions, but had been served daily with fresh meat and bread.[3]

At the close of the Punjaub campaign of 1848-49, the troops located in the country suffered from the disease. They had abundance of fresh meat and bread, of excellent quality, but no fresh vegetables.[4] For some years after stations for troops had been found in the Himalayas, fresh vegetables were not procurable in sufficient quantity. Though the soldiers were provided with good fresh meat and bread, Scurvy was not only present, but was attended with its full mortality.[5]

In the second Burmese war, a detachment at Meanday was dieted for several months on fresh beef in unlimited quantities, biscuit, rum, and rice. The men had, however, no fresh vegetables, nor any substitute for them. After three months, Scurvy made its appearance. Lime-juice was procured and issued freely, and the disease rapidly abated.[1]

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[1] Histoire de Louis IX. par le Sieur Joinville. Bohn's Antiquarian Library.

[2] Dr. Budd, Library of Medicine, art. "Scurvy."

[3] Philosophical Trans. Sir J. Pringle's Address, 1776.

[4] Med. and Surg. Hist. of the British Army, 1858.

[5] Ibid.

Dr. Hammond[2] describes having seen many cases of Scurvy among troops who had fresh-meat rations on four days in the week, and game of their own procuring on the others.

The Turks, as noticed above, in the Mingrelian campaign of 1855-56, had no salt meat, and suffered much more severely from Scurvy than did their French and English allies at a time when the latter were dependent upon it for animal food.

So, also, during the previous winter, "They fed on good sound biscuit, boiled rice, fresh meat twice or three times a week; salt meat was unknown amongst them: they were not overworked or idle, and were in excellent spirits at having beaten the enemy; and yet I found on examination that, on an average, three men out of four on duty in the spring of 1855 were more or less afflicted with Scurvy."[3]

In 1836, above one hundred cases of Scurvy occurred in the 75th regiment whilst quartered in Caffreland, at a time when the men had no harassing duties, and were abundantly supplied with rations of good fresh meat, without having had an ounce of salt provisions. They had no vegetables. The Hottentot troops doing duty with them were served with the same rations, but sought out for themselves pumpkins, melons, some indigenous wild fruits, and esculent roots. They entirely escaped an attack, as also did the 27th and 72d European regiments, encamped at a distance of eighteen miles, fed with the same rations, but supplied also, in addition, with vegetables.

Cases of Scurvy occur every year in North Wales, where fresh meat and milk are abundant, but where the cottagers rear little or no garden produce. Not only the inhabitants, but visitors, located in the country for a short time, are known to suffer, we are informed, occasionally from this disease.

We have ourselves met with many cases of Scurvy amongst the poor of London who had eaten no meat at all for several weeks, but had lived on tea, bread, and butter.[4]

The occurrence of Scurvy so frequently, and in such well-defined form, in cases when salt meat has been absolutely wanting in the dietary, is sufficient to prove that this substance is not a necessary antecedent of the disease, and cannot therefore be properly termed a cause. But it is alleged, sometimes, that it is to the use of salt provisions, combined with the absence of fresh vegetable food, that the disease is owing. If the facts recorded above are insufficient to negative this view, there are two other circumstances which certainly leave the matter in no doubt:—

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[1] Dr. Crawford, Med. and Surg. Hist. of the British Army, 1858.

[2] Mil. Med. and Surg. Essays, p. 192. Philadelphia, 1864.

[3] Dr. Bird, On Scurvy. Lond. 1858.

[4] See also Dublin Med. Press, vol. xviii. Dr. Bellingham; "Observations on the Scurvy," by Dr. Mertans; Phil. Trans. vol. lxviii.; Lond. Med. Gazette, vol. iv. Dr. Shapter; Lancet, 1851, p. 519, Dr. Boyd.

1st, There is no case of Scurvy on record, as occurring in a person who has been adequately supplied with fresh succulent vegetables of good quality.

2d, The occurrence of Scurvy in persons living upon salt meat may be prevented by the regular administration of fresh vegetables, or the juice of lemon.

In connexion with this subject there is an important point to be considered, as regards the relative rapidity with which Scurvy will appear in persons fed upon fresh meat, or salt meat, or who have had no animal food at all. There is great difficulty in arriving at conclusions upon this point. In the crews of vessels, for example, which have quitted a port during the winter, it often happens that a scorbutic taint has been acquired on shore, and before the men were exposed to the limited dietary of ship life. So also on land, before the supply of vegetables is entirely cut off by the failure of a crop, or by the poverty of the patient, a certain amount continues to be taken, although insufficient for the preservation of health. From these and other causes, reliable statistics as to the exact interval which elapses before Scurvy makes its appearance are wanting. The conclusions at which we have arrived, from a careful consideration of recorded outbreaks, as well as from our own personal observation of the disease, are that under a salt-meat diet, Scurvy will appear sooner than when fresh meat has been taken in the ordinary quantity, and that the disease will show itself more rapidly when the patient has been deprived of all animal food, than when he has obtained supplies of salt meat. Liebig has shown that the process of salting deprives flesh of a large proportion of its most important constituents, so that the remainder is deficient in nutritive properties, and the altered and hardened character which it acquires renders even such nourishment as it contains difficult of assimilation. As a result, a diet of salt meat represents only a less degree of starvation than the total absence of animal food, and starvation has been agreed universally to intensify Scurvy. Dr. Kane,[1] the Arctic explorer, speaks highly of the improvement in strength which took place amongst his crew when they succeeded in procuring fresh walrus meat, as a substitute for their salt provisions. He arrived at the conclusion that raw walrus meat was powerfully anti-scorbutic; but his descriptions of the sufferings undergone by his party from Scurvy, even when abundantly supplied with this food, fail completely to justify this opinion. Mr. Whymper[2] expresses an equally favourable opinion of walrus and seal meat, but the details which he gives are not sufficiently elaborate for the purpose of scientific discussion. It seems most probable that fresh meat of any kind, although satisfactorily proved by the instances we have recorded to be incapable of preventing Scurvy, will yet, by its powerful nutritive properties, help considerably to sustain animal strength and retard the development of scorbutic symptoms. So a man, deprived of all food, and dying of starvation, will have life considerably prolonged if he can obtain water. Water, however, we know to represent only a portion of the requirements of man, and to be incapable, by itself, of preserving his existence for more than a short period.

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[1] U. S. Grinnell Expedition. Second Voyage.

[2] Travel and Adventure in Alaska. 1868.

From the limited variety of food which so often accompanies conditions in which Scurvy has appeared, such as besieged towns, encamped armies, and on board ships in long sea-voyages, monotony of diet has been frequently urged by writers as an important cause of Scurvy. But probably one of the most monotonous dietaries in the world is that upon which the poor inhabitants of Ireland thrive,[1] and which consists almost entirely of stirabout, milk, and potatoes. So long as they can obtain this food in sufficient amount, Scurvy is unknown; but when the monotony was broken by the failure in the potato crop, the disease appeared extensively.

The relation, indeed, which such an alleged cause, as well as others which have been most insisted upon—namely, cold and damp, idleness, and mental depression—bears to Scurvy, seems to us simply that which obtains generally in modifications of health. The fact that these conditions are certainly not essential to its production must exclude them from the category of true causes. That they are frequent concomitants, and hasten as well as intensify the symptoms of the disorder, is just what is seen in many other diseased conditions which, depending upon a special cause, are yet capable of being injuriously influenced by circumstances known to affect the assimilation of food, and the consequent nutrition of the animal economy.

SYMPTOMS.—The earliest symptom of Scurvy is a change in the colour of the skin, which becomes pale, sallow, or of a greenish tint, according to the variety of the natural complexion. Succeeding, and indeed often contemporary with this, is a peculiar listlessness of mind, an aversion from exercise, and a condition, not so much of anxiety as to the state of health, as of indisposition to take any trouble regarding it. The patient lounges where formerly he has displayed energy in his occupation; he does not care to speak unless addressed. In reply to inquiries he will usually complain of flying pains about the limbs and back, which he generally refers to rheumatism. The change of aspect, where several individuals are exposed to the same circumstances, will be noticed by them of each other, whilst the observer is unconscious that he, too, is presenting the same appearance.[2] Up to a certain period the appetite remains good, and digestion continues tolerably perfect; usually, however, there is some constipation. There is no fever. Sleep is obtained readily enough. It is sometimes described as accompanied by dreams, in which the luxuries of fruits and vegetables are vividly pictured.[1] Gradually petechiæ are observed especially about the legs and thighs. They are small, of a reddish-brown colour, fading away at the edges, and are especially apt to occur at the points where hairs perforate the skin. They are usually not elevated above the surface. Besides this there may be larger maculæ, apparently formed by the coalescence of several petechiæ, of irregular outline, and particularly common about the lowest part of the legs and on the feet. As the disease advances still larger markings will be noticed, so much resembling bruises as often to be mistaken for the results of violence. Like the eruption of variola, they are apt to fix upon weak portions of the frame for their situation, parts where there has been a blow, strain, or other injury some time previously. Accompanying these external signs there is breathlessness, for which the ear applied to the chest fails to discover any adequate cause. The expression of the countenance is dejected, or it wears an aspect of indifference. The lips are pale. By degrees the face assumes a bloated appearance. In some cases, however, about this period, the eye and its surroundings are the only parts exhibiting signs of Scurvy. The appearance presented is then very remarkable. The integument around one or both orbits is puffed up into a bruise-coloured swelling. The conjunctiva covering the sclerotic is tumid and of a brilliant red colour throughout, " about the eighth of an inch in thickness or elevation above the cornea, leaving the cornea at the bottom of a circular trench or well."[2] There is nothing inflammatory about this condition; it resembles very violent ophthalmia in the colour presented, but there is no pain or discharge. We have seen many cases in which this appearance, together with pallor of the complexion and listlessness, constituted the only evidences of Scurvy, and they have generally been of the most serious character, often terminating fatally. The gums so generally present a remarkable alteration in Scurvy that their condition has been often described by writers as a perfect test of its presence or absence. Our own experience does not correspond with this, and other observers have recorded a similar opinion.[3] All the other phenomena may be present and yet the gums continue in an unaltered condition, except that they are paler than ordinarily. But usually, at an early period of the disease, the gums, first of all pale and contracted, begin to show a swelling at their free margins. This gradually increases so that the teeth are encroached upon, and eventually, in some cases, almost disappear from sight in the huge fleshy masses which encompass them. The swollen gums are then spongy, of a dark-red or livid hue, not sensitive to the touch, and disposed to bleed, sometimes slightly, at others profusely, when irritated. Under these circumstances the teeth become loosened in their sockets, and often fall out. There is a sickening fetid odour from the breath. This is only observed as an accompaniment of the swollen state of the gums, and is evidently due to the sloughing which usually occurs in them. So severe is the affection of the gums in many cases, that fleshy masses like huge granulations are often seen to protrude between the lips. Chewing is completely impossible, and there is some difficulty even in taking fluid nourishment. It is impossible to describe the fearful appearance presented by the sufferer under these circumstances. His skin harsh, dry, dirty-looking, and discoloured with bruise marks, bloated and puffed up in parts by swellings, his whole manner apathetic and helpless, the condition appears to a novice more irremediable than is seen in almost any other disorder. And yet it is remarkable that these cases, where the external manifestations of the disease are so strongly marked, are frequently just those which yield most rapidly and surely to treatment. The change wrought in a few hours by the administration of lemon-juice or vegetables, coupled with general care, is the most extraordinary thing in therapeutics, and of itself furnishes a powerful argument in favour of the cause of Scurvy existing in the absence of such food.

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[1] "The general condition of these populations, although wearing an aspect of great wretchedness to English eyes, is not now unfavourable to health. Throughout the country I found them a fine, well-built, and often athletic race, with children sufficiently fleshy and rosy, and bearing all the marks of health."—DR. E. SMITH, *Sixth Report of the Medical Officer of the Privy Council*, 1863.

[2] This phenomenon is graphically described by Dr. Kane: U. S. Grinnell Expedition.

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[1] U. S. Grinnell Expedition, p. 267.

[2] Bird, On Scurvy, p. 38. See also Dublin Medical Press, vol. xviii. a paper by Dr. Bellingham.

[3] Dublin Med. Press, Dr. Bellingham; "Scurvy in Exeter," Dr. Shapter, Lond. Med. Gazette, vol. iv.; L'Union Médicale, 1857, p. 419: "Scurvy in the French Army in the Crimea," M. Perrin; Dict. de Médecine, art. "Scorbut" (Paris, 1865), 12th edit.

Besides the petechiæ and other larger ecchymoses under the skin which we have described, a most frequent and highly characteristic symptom appears in the occurrence of swellings in the flexures of the joints. A favourite seat of this condition is the ham. The well between the insertions of the flexor muscles is filled up more or less completely by a mass which is hard, but not so unyielding as to be incapable of pitting on pressure. It requires, however, more force to produce this effect than in œdematous swelling, and the impression is retained for a longer period. As this swelling increases, the limb is gradually more and more flexed, doubtless because extension, by stretching the skin over the tumour, is attended with great pain. If the effusion occurs, as it most commonly does, in the lower extremities, the patient is unable to walk. A similar swelling is sometimes noticed at the bend of the elbow, and still more frequently beneath the muscles of the jaw. In the latter position the movements of mastication are very painful. Another common seat of such effusion is under the muscles in front of the tibia, or between the periosteum and that bone where it is subcutaneous. Such tumours have often been mistaken for syphilitic nodes, and mercury has been administered with the result of increasing the scorbutic condition to an alarming extent. It is one of the facts most universally noted, that mercury has a powerfully injurious effect in Scurvy. The skin covering such swellings may retain its colour or present an ecchymosed aspect according as the subcutaneous areolar tissue is invaded or not by the effusion.

The breathlessness which we have noted as an early symptom in Scurvy becomes more confirmed as the disease progresses. On auscultation we find the respiratory murmur louder than natural, but otherwise unaffected. It is very frequently accompanied by occasional faintings, especially when the body is made to quit the horizontal posture. These attacks of syncope are highly perilous. It has happened to us on more than one occasion to witness death from this cause. The patient, previously recumbent, has suddenly sat up in bed to receive our visit, and speedily fallen back in a fainting fit, from which he could not be restored. At Trebizond, notwithstanding that great care was used in the transport of sick from the ships to the hospital, many died whilst being carried up. The danger from fainting is well known at the Dreadnought hospital-ship; and Mr. Harry Leach, the resident medical officer, informs us that no scorbutic patient who is severely affected is allowed to walk up the steps, but is carefully hoisted up the ship's side in a recumbent position.

There is an affection of the chest in Scurvy which, especially when the disease occurs during the prevalence of cold and damp, is very apt to be mistaken for pneumonia. Faint rigors, followed by a certain amount of feverishness, and accompanied by lancinating pain in one or both sides, usher in this condition. The pain is felt only in coughing, and a very viscid mucus is expectorated. The dyspnœa increases, and a constriction as though from a cord bound tightly round the chest is described. Although it occasionally happens that these pulmonary symptoms are dependent upon true inflammation, they are much more commonly associated with effusion of sanguineous fluid into the cavity of the pleura, or into the substance of the lung itself, these structures sharing with every other organ in that tendency to effusion which is the dominant feature of Scurvy. When the lung is thus invaded the expectoration after a short time becomes dark and sanious, with all the horrible fetor which is ordinarily associated with gangrene of the lung, but which is here dependent upon decomposition of the bloody fluid poured into the lung substance. There are now cold sweats, increasing dyspnœa and anxiety, a pulse small and frequent, softer than in inflammatory pneumonia,[1] and death takes place. In other cases there is no pain or cough; but the breathing rapidly becomes short and laborious, and death occurs suddenly. Auscultatory signs of mischief in the lungs are usually wanting; but now and then there is localized dulness on percussion, with bronchial breathing. Or mucous râles are heard; sometimes also gurgling sounds at certain parts of the chest.[2] The symptoms of gangrene of the lung, when it occurs, are indistinguishable from those arising from effusion of fluid which becomes decomposed. The mere occurrence of very fetid and dark sanguinolent sputa is not necessarily an indication of either condition; as its source may exist in the sloughing and bleeding gums. But constant and increasing oppression of the breath, frequent syncope, and great anxiety point unmistakeably to pulmonary mischief; and cases in which these occur are amongst the most hopeless which are ever encountered.

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[1] M. Aug. Haspel, Gazette Médicale, 1859, p. 70.

[2] Haspel, op. cit.

Dulness on percussion may sometimes be noted under circumstances when it probably may be correctly referred to sanguineous effusions into the muscles of the chest, and unconnected with lung mischief. The diaphragm also is sometimes invaded by effusion, and great difficulty of respiration may be thus produced. It is not usually practicable to distinguish the dyspnœa arising from this condition from that caused by lesion of the pulmonary substance, but its significance is not so serious, and it will generally subside with rapidity as the scorbutic state is remedied by dietetic treatment.

As regards the digestive system, the tongue is usually clean and moist. The colour is sometimes red, at others pale, with a violet tinge. It is often large and flabby, showing the teeth-marks at the edges.[1] In the early stages of the disease there is tendency to constipation. Later there is usually more or less of painless diarrhœa, often sanguineous in appearance, but unaccompanied by the other symptoms characteristic of dysentery. Scurvy, when it occurs in camps, is so frequently, if not constantly, complicated with the dysenteric diarrhœa which commonly prevails under those conditions, that disturbances of the digestive system are amongst the most frequent concomitants of the disorder. It does not appear, however, that, apart from the exciting cause, dysentery is to be considered a symptom of the disease. The dejections usually consist of undigested food, with a quantity of colourless fluid somewhat resembling the evacuations of cholera, or they may be accompanied by a considerable flow of dark blood. The slimy, bright, blood-stained, and offensive fæces of dysentery are wanting, unless that disease be present as an accidental complication. A fatal result is not unfrequently due to this exhaustive diarrhœa, the patient becoming worn out by the frequent discharges.

The intellect of patients suffering from Scurvy is usually remarkably free from impairment. The listlessness, however, to which we have referred above, is constantly present, and is sometimes accompanied by great depression of spirits. As a rule, there is complete coherence of ideas, but we have seen cases occasionally in which symptoms of excitement of a maniacal character were present. Ritchie notes this, and describes also tinnitus aurium, muscæ volitantes, vertigo, and deafness as being occasionally complained of.[2]

In confirmed Scurvy the slightest pressure suffices to open the skin and to give rise to an ulcer, whose edges are hard, thick and shining, and the surface fungoid and bleeding. Its tendency is to increase rapidly in size, and to invade the neighbouring structures. An intolerably offensive odour is emitted from it. Ulcers, such as these, will often eat their way into the soft tissues with great rapidity, exposing and invading large vascular trunks, from which dangerous hæmorrhage may occur. Sometimes the disorganization of the flesh is sufficiently complete to expose the bones and produce caries. The lips and nostrils are occasionally the seat of this ulceration, and the patient then presents a ghastly appearance, much like that of an aggravated case of lupus.[1] The exhaustion attendant upon these spreading ulcers is often fatal. Wounds and even slight scratches become invaded by this process. Its influence in cases of frost-bite is most disastrous.

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[1] "There was in general some ptyalism, and then the tongue was indented on its sides, and the swelling of the parotids and of the gums gave the patient the look precisely of a mercurialized person."—Ritchie *On Scorbutus*, Edin. Monthly Journal, July, 1847.

[2] Edin. Monthly Journal, July 1847.

An affection of the sight, to which the title hemeralopia (sometimes also nyctalopia) has been given, is frequently observed in Scurvy. In some cases, recorded by Dr. Bryson,[2] it was the first symptom of the disease noticed. The patients can distinguish objects well enough during daylight, and even at night can read a book held close to a candle, but the moment they pass from the influence of the light they become absolutely blind, and require to be led about. Mr. G. Lawson informs us that several such cases have fallen under his notice at the Moorfields Hospital, but in none has the ophthalmoscope revealed any signs of mischief. The pupils he finds sometimes dilated and sluggish, in other cases natural. The condition rapidly subsides under an anti-scorbutic regimen. Mr. Soelberg Wells has met with the symptom in cases of great depression of the nervous system, after severe illnesses, and in badly-fed and cachectic subjects. It sometimes occurs in prisons. "It ought," he writes us, "to be carefully distinguished from that which depends upon retinitis pigmentosa, in which the ophthalmoscopic appearances are most marked, which is not amenable to treatment, and generally leads, sooner or later, to almost, if not complete, blindness."

DIAGNOSIS.—In typical cases of Scurvy, and especially when, from circumstances, the occurrence of the disease is probable, the diagnosis is very easy. The dirty pallor and bloated condition of face, remarkable lethargy and indisposition to exertion, ecchymosed state of the skin generally, with contraction of the limbs from effusions, and spongy bleeding state of the gums, form a combination of symptoms which readily distinguishes Scurvy from every other disease. But in the commencement of an outbreak, in sporadic cases, or when the attack is slight, the nature of the ailment is very often mistaken. The patients who present themselves for medical assistance complain of weakness, of pains in the limbs and back which are almost always referred to rheumatism, or of pain in the stomach. They say nothing probably of the state of the skin covering their legs, or of any sponginess of the gums, so that the condition of these structures is not perceived by the attendant, who applies himself accordingly to the relief of the symptoms detailed to him by the patient, and the true nature of the case is consequently overlooked. The colour of the skin (especially when dirty) in ordinary chlorosis, strongly resembles that of a scorbutic patient, and in such a case there is often a dull heaviness of manner which might tend to mislead an observer. In fact, the early stage of Scurvy is really a form of chlorosis, produced, however, by special circumstances, and remediable only by a correction of these. A careful examination of the skin and gums and the history of the illness will serve to distinguish the conditions.

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[1] Bird, On Scurvy, p. 9.

[2] Ophthal. Hosp. Reports, July 1859, p. 40.

The red and purple spots, livid blotches, and bruise-like stains which occur in purpura, and closely resemble those found in Scurvy, may possibly cause some hesitation in diagnosis. But there is little difficulty in distinguishing the two disorders. In purpura these appearances often present themselves suddenly in a patient previously in fair health. This is never the case in Scurvy. The latter disease is always gradual in its progress, and it will be found, on inquiry, that a period of increasing pallor, debility, and listlessness, preceded the appearance of petechiæ or blotches. The very peculiar dirty pallor of complexion, so characteristic of Scurvy, is absent in purpura. So also is the tendency to effusions about the joints, causing contraction of the limbs, and the spongy and bleeding gums. Lastly, the occurrence of purpura is entirely independent of the defect in diet which produces Scurvy, and it is not cured by fresh vegetable juices. There would never, indeed, be any difficulty in the diagnosis of Scurvy but for its comparative rarity, at least in an advanced form, in civilized life. Under circumstances of privation, as in winter campaigns of armies, Arctic expeditions, and the like, the disease is usually expected and recognised as a matter of course. Not so, however, in ordinary civil practice. Amongst the classes which furnish the out-patients to our hospitals, dispensaries, and unions, pallor and debility are more often present than absent. Even bruise-marks from accidental injuries are not at all uncommon, so that unless the possibility of Scurvy be pretty constantly remembered, and the symptoms be tested by a reference to that disorder, there is great probability of a number of cases being overlooked. There would be less liability to such errors if the alteration of the gums commonly attracted the patient's attention, and was expressly pointed out to the medical attendant. This very rarely happens. In persons of the class referred to the teeth are rarely or never brushed, and the gums, consequently, are often in a more or less unhealthy condition, so that a little additional discomfort in this respect is scarcely regarded. It is considered a trivial matter in comparison with the so-called rheumatic pains from which they suffer, and for which alone they ask relief. Were scorbutic patients voluntarily to tender a history of all their symptoms, and the dietetic conditions under which they have been living, there could be little chance of any instructed person, even though he had never seen the disease, coming to a wrong conclusion. But this, it may safely be said, never occurs.

The complaints made by the patients of debility and pains in the limbs are just those symptoms of the disorder which are the most likely to be referred to other causes, and it is needful, therefore, that the medical attendant should himself institute the inquiries necessary to prove the presence of Scurvy in his patient. This, of course, he fails to do unless an idea of the probable nature of the disease has presented itself to his mind. We are induced to dwell upon this point from a conviction that there are still many members of the profession who, because no patient has ever consulted them for a swollen and bleeding state of the gums, are under the impression that they have never met with a case of Scurvy—a conclusion, it will be seen, by no means well founded.

PATHOLOGY.—Great obscurity still involves the question of the ultimate cause of Scurvy. That the proximate cause is an alteration in the quality of the blood, induced by the absence from the ingested food of fresh vegetable juices, is sufficiently manifest from the history of the disease. But we are still in the dark upon three points of importance—

1st. What is the essential element contained in fresh vegetable material by the deficiency of which in these cases such remarkable changes are produced?

2d. Is the influence of this element exerted upon the chemical or the physical quality of the blood?

3d. By what physical law does the blood so altered in quality exhibit such changes in its relation to the tissues?

The essential feature of Scurvy, upon which is based nearly the whole series of organic lesions which takes place, is this,—that the relation between the blood flowing in the capillaries and the tissues is so altered as to permit of the diversion of some or all of the blood constituents from their natural receptacles into tissues from which they are excluded in a state of health. Beyond this, however, there are the cases of fatal syncope, which do not admit of such an explanation. In such instances it would seem, either that the muscular structure of the heart is so weakened by mal-nutrition as to lose the power of efficient contraction, or, what is still more probable, the phenomenon is due to embolism.

It is impossible to offer any certain solution to the second and the third questions. Owing to the absence of a perfectly satisfactory mode of analysis of the blood, not only in cases of Scurvy, but in a state of health, no theory can be safely based upon such accounts of the alterations manifested by scorbutic blood as have been published. The only positive modification that can be detected in the blood, is a very considerable diminution in its density.[1] It is quite conceivable that the exudations of sanguineous fluid may depend upon the increased tendency to exosmose which such an alteration would necessarily produce.

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[1] MM. Becquerel and Rodier, Lancet, 1847, vol. ii.

With regard to the first question, we are able to arrive, principally by a process of exclusion, at something which probably approaches the truth. We have seen that Scurvy will occur where there is no deficiency in the albuminous, oleaginous, or saccharine elements of food, but where there is a want of something which fresh vegetables can alone supply. Fresh lemon-juice may be taken as a convenient instance of a material which is able to prevent the development of Scurvy, and to cure it if it has already appeared. Lemon-juice contains free citric acid, mucus, vegetable albumen, and sugar,[1] with small quantities of malic acid, and acid salts, especially of potash.[2] The only ingredients which it is necessary to consider attentively in this analysis are citric and malic acid, and potash. The other substances are found abundantly in food which is not anti-scorbutic.

In 1848 Dr. Garrod brought forward a very ingenious view of the cause of Scurvy, which has attracted much attention.[3] From examinations of food under the use of which Scurvy was capable of occurring, he was led to the conclusions, "that in all scorbutic diets, potash exists in much smaller quantities than in those which are capable of maintaining health," and "that all substances proved to act as anti-scorbutics contain a large amount of potash." It is, then, to the absence from the food of a requisite amount of potash that he attributes the occurrence of Scurvy, and it is to the presence of potash in lemon-juice that he ascribes the anti-scorbutic power of that material. But for his argument to have been complete, the converse of his second proposition should have been true also. It ought to have been shown that all substances largely containing potash are anti-scorbutic. This is not the case. By reference to Dr. Garrod's table of analysis, we find that one ounce of boiled mutton contains 0·637 grain of potash, whilst one fluid ounce of lemon-juice contains but little more, 0·846 grain. So that two ounces of boiled mutton added daily to a dietary under which Scurvy is occurring should be even more efficacious than one ounce of lemon-juice. The numerous instances on record (to some of which we have referred) of fatal Scurvy occurring in persons abundantly supplied with fresh meat, and on the other hand the indisputable power of lemon-juice in preventing the disorder, are sufficient to disprove this. Again, in the records of Scurvy disasters on board merchant vessels, pea-soup always figures as a most important part of the dietary of the crew. They have often been driven to subsist almost entirely upon this food, owing to the bad quality of the meat. One ounce of peas, according to Dr. Garrod's table, contains 0·529 grain of potash—a large proportion, considering that in an ounce of white flour only 0·1 grain is found. Yet peas are well known to be utterly useless in the prevention or cure of Scurvy.

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[1] Witt. Chem. Soc. Quart. Journal, vii. p. 44.

[2] Garrod, loc. cit.

[3] Edin. Monthly Journal, January 1848.

The crucial test of administering nitrate of potash to Scurvy patients has been applied, and found wanting.[1] Moreover, Dr. J. O. Grant describes the occurrence of Scurvy amongst the Ottawa "lumberers" living upon pork salted with nitrate of potash. In one shanty he found twenty-five men out of thirty-six attacked with the disease.[2]

We are thus led to the conclusion that it is either to the free organic acids which exist in lemon-juice, or to the acid salts, that the efficacy of this material is owing. Now, the influence of the citric acid of commerce in Scurvy is by no means certainly proved. Statements and opinions on this point are very contradictory, but the bulk of evidence is certainly opposed to the utility of citric, as well as of tartaric and acetic, acid. There seems reason to believe that the bitartrate and citrate of potash have some influence as anti-scorbutics, though their power is certainly far less than that of fresh vegetable juices. It is probable therefore that although the organic acids and potash separately do not represent the requisite material, it is to be found in the chemical combination of the acid and base. Very possibly the form in which these salts exist in lemon-juice renders them more easily absorbed and decomposed by the digestive organs, than when exhibited separately. The Materia Medica gives numerous analogous examples of the superior efficacy of a medicine in its natural combinations.[3] No artificial imitation of mineral waters is equal to the supply from their natural source. All anti-scorbutic juices contain salts of citric, tartaric, or malic acids, and we have no evidence of any substances which contain these materials in considerable quantity, and are yet deficient in the power of preventing Scurvy. The mode by which they act is still involved in obscurity.

Morbid Anatomy.—The body of a patient who has died of Scurvy is generally emaciated, but this is by no means always the case. Where the diet has been absolutely deficient, or of such a nature that its mastication was almost impossible from the condition of the gums, there is much wasting. But Scurvy, as we have seen, may occur when there has been not only no lack of food, but the nutriment has been of a kind easily taken by the patient, although from its quality it has not been able to prevent the disease. Under such circumstances the general bulk and weight of the body are preserved, whilst the tissues are found to present the appearances characteristic of the disorder.

Externally, the body presents the same general aspect as was observed during life. Decomposition is more than ordinarily rapid. The extremities are usually rigid. Blood is sometimes observed to flow from the mucous passages. Blood, or fibrinous effusion, more or less strongly blood-coloured, is found extravasated under the skin, into the subcutaneous areolar tissue, and into the aponeurotic sheaths of the muscles, sometimes bruising and breaking the muscular fibres. The lower extremities, and especially the hams, are generally the most severely affected, but the same condition may be found in the arms, particularly about the bend of the elbow, and under the pterygoid muscles of the jaw. These effusions, when they take place, under the periosteum, sometimes lead to death of the bone beneath, and this has not unfrequently occurred in the jaws. Simple serous effusions, besides, depending apparently on the obstructed circulation, occur, especially about the feet and ankles, so as to give a peculiarly clumsy appearance to the lower extremities.

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[1] See Med. Times and Gazette, vol. xx. Dr. Murray; Med. Times, March 23, 1850, Dr. Bryson, R.N.

[2] Med. Times and Gazette, December 26, 1863.

[3] Parkes, quoted by Aitken.

The condition of the brain varies considerably. It is often free from any appearance of lesion. Sometimes there is effusion of serum under the arachnoid and into the ventricles, whilst the vessels on the brain surface are empty, and the general aspect of its substance is pale. In other cases the cerebral vessels are gorged with very dark fluid blood, or coagula; and there may be ecchymoses upon the surface of the brain, and sanguineous effusion into its substance.

Serous fluid, sometimes in large quantities, is frequently found in the pleural cavities. The lungs may be pale, shrunk, and bloodless in appearance, or gorged with serous fluid, and, sometimes, with very dark blood. When grave symptoms of mischief in the chest have presented themselves during life, stains, or violet marblings, like to those on the skin, have been found upon the surface of the lungs after death. On cutting into these they are found to be of varying depth, but usually superficial. Internally, the vesicles and small bronchi contain a muco-sanguinolent product; there is, besides, slight or severe bloody infiltration into the cellular interstices of the pulmonary vesicles, occupying especially the bases of the lungs, and characterised by a red-winey tinge, with impregnation of black blood. In certain cases the lung offers in some points all the characters of the most complete engorgement, loss of elasticity, crepitation, and permeability, increase of volume and of weight. These characters, however, are never carried to the extent seen in ordinary pneumonia. It is principally at the diaphragmatic aspect inferiorly and posteriorly that this engorgement is seen; physical laws tending materially to the choice of this site. Little deposits of blood, not coagulated, are found in different parts of the lungs. These are of variable volume, and constitute, in fact, a species of ecchymotic collections, which compress and obliterate little by little the pulmonary vesicles. They form sometimes largish, fluctuating tumours, composed of liquid blood without clots, contained in cavities of irregular form, which are not lined by any membrane. The sudden rupture of such tumours causes considerable hæmoptysis. Occasionally the lung will be found gangrenous; it is then characterised by the usual greenish-grey colour of its structure, mixed with darkened fragments, and imbued with air-bubbles and an ichorous bloody liquid, breaking up under slight pressure, and emitting a most offensive odour.[1]

An equal uncertainty attends the condition in which the heart may be found. It is sometimes pale and flaccid, with the cavities quite empty. In other cases it is filled with black liquid blood, and its cavities are dilated. Occasionally its lining membrane, as also that of the aorta and pulmonary artery, is stained with a reddish tinge.[2] Black fluid blood is sometimes found in the pericardium. The muscular substance of the heart may be ecchymosed.

The muscular and mucous coats of the stomach and intestines are invaded usually by sanguineous effusions, and the deposits present all that variety of colour which is characteristic of bruises in their various stages, varying from a pink to a blackish-green tinge. The intestines themselves may contain fluid blood. Dr. Ritchie describes an enlarged condition of the solitary glands in the lower part of the ileum. The mucous surface of the intestine is sometimes abraded, with minute and superficial ulceration, or it may present detached black ulcers of considerable size. There is a tendency to increase of severity in these towards the lower extremity of the bowel.[3]

The liver and spleen are often, but by no means always, enlarged, gorged with dark blood, and their structure softened and friable.

With all the variety which may present itself in the post-mortem appearances, there is one appearance which is constant in all cases of death from Scurvy. In some part or other, sanguineous effusion into the tissues will be discovered. Considering the delicate structure of the brain, it is remarkable that lesions of this organ occur by no means so commonly as in other and less vital parts of the economy.

As regards the nature of the effusions which play so important a part in the fatal results of Scurvy, minute observations would seem to show that they are essentially fibrinous in character, more or less coloured by blood-corpuscles. They are sometimes gelatinous in consistence, marked with streaks of a pale-yellow colour, somewhat resembling the fibrinous clots so often seen in the heart. They occasionally exhibit a higher degree of organization. Deposits of this kind occur in the form of layers of from a quarter of a line to a line in thickness, composed, apparently, of fibrin of a bright yellowish-red colour, firm and elastic, affording no fluid on pressure; they are, in fact, false membranes, and are quite distinct from the surrounding muscles, to which they adhere. This kind of deposit has been termed "scorbutic formation." The stiffness of the joints, and especially of the knees, appears to be caused by the firm consistence of these effusions.[4] Upon injection, capillary vessels have been discovered, which in their character and mode of distribution are similar to those met with in other recently organized adventitious tissues. It seems most probable that the hypertrophy of the gums proceeds from the deposit of a plastic material. They are firm, and bleed when wounded. The rapid absorption which they undergo upon treatment is inconsistent with the supposition that they are distended with blood, but is explicable upon the view of the fibrinous character of the enlargement.[1]

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[1] Gazette Médicale, 1859, p. 70: M. Haspel. The description of the morbid appearances in the lungs is mainly derived from M. Haspel's minute and graphic account.

[2] Ritchie, loc. cit.

[3] Ibid.

[4] Dr. Himmelstiern, Brit. and Foreign Med. Rev. vol. xx. p. 150.

PROGNOSIS.—When a patient affected with Scurvy is placed under favourable circumstances as regards food, shelter, &c., his recovery may be safely expected, provided that irreparable mischief has not already occurred in organs essential to life. It is remarkable that the severity of the external manifestations of Scurvy is by no means an accurate guide to a safe prognosis. The fact has been noticed by many observers, and we have ourselves repeatedly witnessed it, that when the patient, from the extent to which his skin, muscles, and gums were involved, appeared most severely affected, recovery would very frequently be much more certain and rapid than in cases in which such serious external signs were absent. Were it not inconsistent with what we know of the pathology of the disease, it would appear that a degree of safety to internal organs was procured by this severe affection of the outside of the body; as though, indeed, the fury of the disorder expended itself on parts of the economy the least essential to life. In general terms the prognosis will be favourable, or the reverse, according to the degree to which such structures as the brain, lungs, and heart have escaped serious lesion, or are manifestly much involved.

THERAPEUTICS.—The treatment of Scurvy almost entirely consists in supplying the patient, in the most easily assimilable form, with that material by the deficiency of which his disorder has been produced. Combined with this there will, of course, be needed such a judicious arrangement of general diet as will most easily contribute to his general nutrition. The choice of this will much depend upon the condition of the patient's gums and digestive organs. It is very important that his diet should be varied as much as possible, consistent with the avoidance of diarrhœa. Fresh lemon-juice, in the form of lemonade, should be administered as the ordinary drink, *ad libitum*. The existence of diarrhœa should be no reason for withholding this treatment. The looseness of the bowels in Scurvy will be uninfluenced by any medical appliance, so long as the scorbutic condition of the blood remains uncorrected; and the fresh juice of the lemon has been proved to be more easily digested than any other form of vegetable food. Professor Maclean[2] speaks highly of the use of the Bael fruit in the dysentery which is often associated with Scurvy. The fruit contains a large quantity of tannin with vegetable mucus, a bitter principle, and a vegetable acid. It is much used in Bengal. Professor Maclean has seen it useful when all other measures have failed. According to circumstances, the food may consist besides of good beef-tea, with eggs beaten up with wine, or, if the patient can bear it, solid fresh meat roasted or boiled, mashed potatoes, cabbage, milk, salad, or sauer-kraut. The diet will require careful observation; but the great general principle is to be borne in mind that the anti-scorbutic principle must be received by the patient in one form or other if his treatment is to be successful. When diarrhœa is persistent, the trisnitrate of bismuth with opium may often be given with great advantage, the use of fresh lemon-juice being, however, continued at the same time. When the gums slough and bleed very much, they should be brushed over daily with solid nitrate of silver. An amount of relief is thus afforded which it is difficult to explain. Our Turkish patients were so alive to this that they used to indicate to us by pantomime their desire to have the application repeated at every visit. In effusions under the periosteum the iodide of potassium has been found very useful in relieving pain and hastening absorption.[1]

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[1] See also Budd on Scurvy, Lib. of Med. vol. v. p. 86.

[2] Aitken's Science and Practice of Medicine, vol. i. p. 624.

For the offensive fœtor of the breath, washes of chlorine may be used, and probably Condy's fluid freely diluted would be a good application. For the hard swellings in the hams and legs, friction with warm soapsuds and water, several times a day, was employed with most success in the Turkish hospitals. Scorbutic ulcers may be dressed with lint steeped in lemon-juice and covered with oil-silk. But all local remedies are but palliatives, and are inert as substitutes for the constitutional treatment of the disorder.

Amongst the vegetables which may be used as preventives of Scurvy are oranges, lemons, limes, cabbage, lettuce, potatoes, onions, mustard and cress, dandelion, sorrel, scurvy-grass, the *Agave americana* (cactus), grapes. An ounce of lemon-juice should be issued daily, when vegetables run short, and, as on board ship, should be begun ten days after the deprivation of vegetables.[2] Potatoes may be conveniently preserved in casks, with the addition of molasses.

Of late years a very ingenious form of preserved vegetables has been prepared by MM. Masson and Chollet, of Paris. It consists of cauliflower, carrot, lettuce, peas, &c. dried and compressed into solid slabs, which are very portable, and keep good for a length of time. The preparation requires soaking for four or five hours in water, before use, and then should be cooked very slowly. The experience of the Crimean and American wars has shown it to be convenient and palatable enough; but, as a preventive of Scurvy, far behind the fresh vegetables or lime-juice in efficacy. It may be added to these with advantage, but cannot replace them. Dr. A. Marroin,[3] in his record of Scurvy in the French fleet, says, "Preserved vegetables retard the outbreak of Scurvy: they slacken its march without stopping it altogether, when it has thrown its roots deeply into the economy." They were liberally supplied to the crews of the French ships in the Black Sea during 1855. Yet, in the second quarter of the year, thirty cases of Scurvy are recorded, whilst in the third quarter (the summer months) no less than 531 cases occurred. In October, 427 cases are mentioned. "In November, during the first fifteen days, we numbered more than 1,000 scorbutics on the six vessels in Katcha Roads."[1] One vessel alone, the *Friedland*, had no less than 400 cases on board. Some of the ships were then sent down to the Bosphorus, with all the scorbutics on board. On their arrival, the mess-tables were well supplied with salads, fruits, &c. "The change effected by this was instantaneous. Those who presented symptoms of Scurvy, saw them disappear; those (and they were even still more numerous) who experienced that muscular debility and horror of movement which constitute the warnings of it, were, after a few days, restored to perfect health."

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[1] Bird, On Scurvy, p. 16.

[2] Parkes, Practical Hygiene, p. 248.

[3] Histoire Médicale de la Flotte française, p. 104. Paris, 1861.

In the Confederate army (Dr. Darby informs us) the yam, which is generally cultivated throughout the South, was found very beneficial. Syrup from the Chinese sugar-cane (sorghum), abundantly manufactured in the last two years of the war, was issued as a ration with decided benefit. When badly made it is liable to fermentation, and deranges the bowels, yet even in this condition it was of great service in scorbutic cases, as was also the extracted juice before being boiled.

Apples certainly possess anti-scorbutic properties, but they are inferior to lemons and oranges. Cider would appear to be deficient in the property. Dr. Boyd[2] relates two cases of Scurvy which occurred in lunatics, who had lived for several weeks exclusively upon bread and cheese, three times daily, with a pint of cider at each meal!

Sauer-kraut has long been recognised as very efficacious. It was employed by Captain Cook with signal advantage.

It is probable that the light French wines possess considerable anti-scorbutic power.

It is not often that the power of milk, as an anti-scorbutic, is able to be tested in adults, but the immunity from Scurvy of infants subsisting entirely upon this food shows that it possesses the property to a very large extent. We have lately had under our observation an adult patient, who, in consequence of organic disease of the pylorus, was unable to take ordinary diet, and who subsisted for seven months upon Oswego boiled with milk, without any symptoms of Scurvy occurring.

As regards the preservation and use of lemon-juice, the following practical suggestions have been recently issued by the Board of Trade for the information of shipowners and shipmasters:—

"Every ship on a long voyage should be supplied with a proper quantity of *lime* or *lemon juice*.

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[1] Marroin, loc. cit.

[2] Lancet, 1851, vol. i. p. 519.

“The juice having been received in bulk from the vendors, should be examined and analysed by a competent medical officer. All measures adopted for its preservation are worthless, unless it be clearly ascertained that a pure article has been supplied.

“Ten per cent. of brandy (sp. gr. 930), or of rum (sp. gr. 890), should afterwards be added to it.

“It should be packed in jars or bottles, each containing one gallon or less, covered with a layer of oil, and closely packed and sealed.

“Each man should have at least two ounces (four table-spoonfuls) twice a week, to be increased to an ounce daily if any symptoms of Scurvy manifest themselves.

“The giving out of lime or lemon juice should not be delayed longer than a fortnight after the vessel has put to sea.

PURPURA.

By Thomas Hillier, M.D.

Revised by Tilbury Fox, M.D.

The term Purpura is derived from *purpureus*, purple, and, as generally used, it signifies a discoloration of the skin, of small size, due to the occurrence of hæmorrhage into its substance, and unaccompanied by any serious disturbance of the general health, or any appreciable organic disease. The hæmorrhage into the skin is, in fact, the disease. Sometimes, however, the term is applied to cutaneous hæmorrhage occurring in diseases in which the general condition is the more important, as in "purpuric" fever.

The occurrence of hæmorrhage in the cutis, in fact, from whatever cause, has been considered sufficient to warrant the use of this term. Cutaneous hæmorrhage is often seen in typhus fever, in measles, in small-pox, in scurvy, and as a result of injury, as well as in cases known as Purpura proper. Typhus fever has been described as Purpura contagiosa, but this name is now obsolete; the cutaneous hæmorrhage which often accompanies it being properly regarded as a symptom of secondary importance. In scorbutus or scurvy we have another well-defined disease, in which hæmorrhage into the skin constitutes an important symptom. Purpura has been, and often is, confounded with scurvy, but should be carefully distinguished from it. With the advance of medical knowledge it is very likely that the cases even now classed together under the name of Purpura, will be further distributed under several distinct categories according to their real pathological character. It might be well to use the term cutaneous hæmorrhage for extravasations of blood connected with general diseases, or violence, and to limit the use of the term Purpura to those forms in which the skin alone seems to be at fault.

Willan described four varieties: Purpura simplex, P. hæmorrhagica, P. urticans, and P. senilis, and classed the disease with the exanthemata. Purpura, however, is readily distinguished: the patches are not transitory, and do not enlarge, they undergo absorption, changing colour day by day, like a bruise; the colour of true exanthems disappears on pressure, the blood still remaining in the vessels, whilst in Purpura the colouring matter, and sometimes the blood-corpuscles, are out of the vessels, and the stain is, therefore, not effaced by pressure.

In the present day, however, Purpura, in its more limited and proper rendering, is commonly described under two main divisions: Purpura simplex, and Purpura hæmorrhagica. In Purpura simplex, hæmorrhage is confined to the skin; in Purpura hæmorrhagica blood escapes also from the mucous surfaces, the alimentary, the genito-urinary, and the respiratory.

Purpura urticans is a compound disease, made up of urticaria and Purpura. Purpura senilis is merely the disease Purpura in old people, in which the patches are large.

SYMPTOMS.—On the skin, a number of spots or patches make their appearance; these vary in size from mere points, when they are called *stigmata*, or the size of pins' heads or peas (*petechiæ*), to large patches of considerable dimensions, even several inches in diameter. The term *vibices* is applied to the patches whose length much exceeds their breadth, so that they resemble the bruise left by a whip or stick, and *ecchymosis* is the name employed when their shape is more irregular. The colour varies from a bright red, especially at the early stage of the disease, to a violet, deep purple, or blackish tint. When the eruption first appears, the spots generally have an abrupt, well-defined margin; but as they fade, their outline is gradually lost in the surrounding skin. Occasionally in the early stage, if examined with a lens, the outline may be irregular. In some cases, the discoloration of the skin is preceded by subcutaneous swelling and induration. The discoloured spots are not themselves raised. Occasionally, there is an escape of blood-stained fluid under the cuticle, giving rise to blebs on the affected patches. Some writers express this fact in other words, by stating that Purpura is complicated with pemphigus. When the hæmorrhage infiltrates the skin very closely, the derma is found, on the removal of the cuticle, to be thickened, hard, and of a purple colour. It speedily dries, and has lost its sensibility; it is, in fact, dead. Aggravated cases of this kind are of rare occurrence.

Spots of Purpura are distinguished from simple exanthems by not disappearing or fading on pressure. This is a symptom common to all cutaneous hæmorrhages. In a few days the eruption of Purpura changes colour, passing through various shades of red, orange, and yellow, until it completely disappears. Whilst some spots are fading, fresh ones appear, or successive eruptions may occur, separated by intervals in which no eruption is seen. Together with the cutaneous phenomena, hæmorrhages often occur from various mucous surfaces, giving rise to epistaxis, hæmaturia, melæna, hæmatemesis, or hæmoptysis. It might be said that Purpura simplex, when severe, may be accompanied by internal hæmorrhage, and then passes into Purpura hæmorrhagica. This will be noticed in speaking of Purpura hæmorrhagica.

VARIETIES OF PURPURA.

Purpura Simplex.—Purpura in its mildest form is commonly seen on the legs in the form of small bright-red spots, which make their appearance without any other symptom beyond slight lassitude, and, in some cases, aching of the limbs. The first eruption soon disappears, but is often speedily followed by a second, and this again by a third. Its appearance is favoured by standing, and the cure is promoted by rest, and by the use of elastic bandages. An attack of this kind often occurs during the convalescence from rheumatic fever, and in persons whose general health is impaired from any cause. A special variety, Purpura rheumatica, has been indeed named by Schönlein. In this form of the disease there are antecedent pains about the body, especially the knees; the spots are freely and widely developed, and have a border oftentimes of a somewhat bright hue; they are of the size of hemp seed, or thereabouts. A fresh crop and fresh pains go together. This variety is observed to occur in the strong; it is most frequent in those between the ages of twenty and thirty; it is never seen in children, and is more frequent in males than females. It is only Purpura occurring in rheumatic subjects.

Purpura senilis is a form of cutaneous hæmorrhage especially frequent on the upper extremities of old women whose arms are much exposed to weather and local irritants. The degenerate state of the tissues of the old predispose to its occurrence, and the extravasation of blood often follows more than ordinary exertion or direct injury of the skin, or undue pressure of some kind. Bateman thus describes it: "It appears principally along the outside of the forearm, in successive dark purple blotches of an irregular form and various magnitude. Each of these continues from a week to ten or twelve days, when the extravasated blood is absorbed. A constant series of these ecchymoses had appeared in one case during ten years, and in others for a shorter period, but in all the skin of the arms was left of a brown colour." It is not attended with any constitutional disturbance.

In diseases of the heart causing obstruction to the capillary circulation, petechiæ are occasionally seen. Five interesting cases of the kind are to be found in the Trans. Path. Society, vol. xvii., 1860, p. 422, by Dr. Ogle. In one case the purpuric spots vanished whenever the patient kept his bed. If he were allowed to get up and go about, the spots would again appear.

Purpura Hæmorrhagica.—So far as the skin is concerned, the phenomena are the same as in Purpura simplex, except that the morbid appearances are commonly more aggravated. The term "hæmorrhagica" is given when there is, in addition to cutaneous hæmorrhage, a flow of blood from the free surface of mucous membranes. The most common hæmorrhage is epistaxis, which may be slight in amount or very profuse. It occurs alone or in connexion with hæmorrhage from other mucous surfaces. Hæmatemesis is of less frequent occurrence; it is sometimes very profuse; melæna also occurs, from hæmorrhage either into the large or small intestines. Bleeding may take place from the gums, but is not nearly so frequent as in scurvy. Besides the blood which escapes from the free surfaces, there is sometimes extravasation into the substance of the mucous membranes: this may be often seen on the palate, inside the cheeks, or on the gums. Small blisters containing bloody serum are sometimes found on the tongue and other parts of the mouth; these soon break, and their contents escape.

Hæmorrhage into the conjunctiva is not uncommon. Hæmaturia is a frequent symptom; the blood may come from the pelvis of the kidneys, the ureters, or the bladder. Hæmoptysis, dependent on hæmorrhage from the bronchi, is not so often met with. Menorrhagia and hæmorrhage from the external auditory meatus are occasional symptoms. It is not often that extravasation occurs into the parenchyma of organs; it has, however, been seen in the brain and lungs; and death has resulted from cerebral or pulmonary apoplexy. Blood is sometimes extravasated into the substance of the muscles. Purpura hæmorrhagica, like the simple form, is either ushered in by fever or occurs quite independently of febrile disturbance. It may occur in the midst of apparently good health, or it may be preceded for some weeks by great lassitude, faintness, and pains in the limbs and joints. It is usually accompanied by a sensation of great debility and depression of spirits. The pulse is generally weak, and may be quickened; it is sometimes, on the contrary, full and not frequent; occasionally it is intermittent. Paroxysms of fever resembling hectic have been sometimes observed. As the disease advances, signs of anæmia manifest themselves, great pallor of the mucous membranes, a venous hum in the neck, tinnitus aurium, and shortness of breath. Œdema of the face and feet may sometimes occur. Some patients are liable to attacks of slight faintness or complete syncope. The digestive functions are in some cases quite unaffected; in others they are more or less disordered. There may be tenderness in the epigastrium, with a sensation of fulness, nausea, and either constipation or diarrhœa. Deep-seated pains in the abdomen, chest, or back are also sometimes present; these are either caused by congestion of the liver, lungs, kidneys, or other viscera, or are merely of a neuralgic character. The urine sometimes contains blood; at other times, casts of the tubes and albumen. This condition may first appear and subside with the cutaneous phenomena, or Purpura may occur in the course of chronic Bright's disease, or the renal changes may follow Purpura. The duration of the disease is very various, from a week to several months.

Dr. Graves, in his Clinical Medicine (vol. ii. p. 362), describes two cases having some of the characters of Purpura hæmorrhagica, but presenting important points of difference; he proposes for them the name of "Exanthema hæmorrhagicum." There was an eruption of red spots, somewhat resembling that of measles, but without the crescentic outline usually seen in this disease, and more nearly resembling that of typhus. The patients were strong and healthy previous to their attacks. There was but slight febrile excitement, and this only at the onset; the pulse was slow (in one case 70, and in the other 50 in the minute), but it was peculiarly hard and thrilling, almost dicrotous. There was no headache, delirium, or loss of sleep. Bleeding took place from the intestines and urinary organs, from the gums, the nares, stomach, and other mucous surfaces; it gradually became more profuse, resisting all treatment. The tongue was dry and brown in both cases. The exanthems appeared on the skin on the seventh day of illness, in one case, and after seventeen days in another; the spots never became petechial, and disappeared in about five days. Both cases proved fatal in about four weeks. In one case the eruption was ushered in by a tingling sensation, resembling the sting of nettles, and was diffused over the trunk and extremities; in the other, there was no such tingling, and it was limited to the limbs. One man, aged 34, had been a great spirit-drinker, and the symptoms commenced immediately after drinking cold water, whilst in a state of profuse perspiration. The other man, aged 29, was of temperate habits, and no probable cause could be thought of. The cases appear to have been like acute Purpura as regards the mucous membranes, but unlike it in presenting an exanthem but no hæmorrhage in the skin. Whilst the pathology of Purpura is so imperfectly understood, these cases may be classed with it; hereafter, they may be more clearly distinguished from it. Dr. Graves states that, if cases of this kind should again come under his notice, he would freely resort to blood-letting, as affording the best chance of cure.

Dr. Lingen (in the British Medical Journal, July 27, 1867, p. 61) refers to a group of cases of "Irish Purpuric disease." A pig was taken ill, it was drowsy, "stiff," and before death exhibited purpuric spots on its belly, face, and nose. A servant girl who attended to the pig, was attacked with febrile symptoms, and was ill a few weeks. The owner of the pig had suffered, and had large purpuric spots on the loins, the thighs, and smaller ones in other parts. The cowman died in three days of a similar attack. "Purples" in pigs are common in America.

Purpura urticans is a complication of abortive urticaria with Purpura simplex. It is characterised by "rounded and reddish elevations of the cuticle, resembling wheals, but which are not accompanied, like the wheals of urticara, by any sensation of itching or tingling." They gradually form and subside within twenty-four or thirty-six hours. They are usually seated on the legs, and are interspersed with petechiæ; they are also seen on the thighs, breast, and arms. Whilst some spots fade, others appear in succession for three or four weeks. This form of disease is not usually attended with fever; it is most common in warm weather; it is often accompanied by œdema, and some stiffness and pain in the legs. It has affinities with erythema tuberculatum.

This variety of Purpura illustrates an important clinical fact, viz. that cutaneous hæmorrhage may occur, under certain circumstances, at the seat of ordinary eruptions, accompanied by congestion; the changes in the tissues predisposing to the escape of blood from the vessels. Under these conditions, the eruption which is supplanted by the hæmorrhage is abortive. The writer has seen, for instance, a purpuric patch take the place of a cluster of vesicles in zoster. The same thing is seen sometimes in erythema, and in connexion with lichen, constituting *lichen lividus,* which Hebra describes under the term *Purpura papulosa.*

A form of Purpura attended with pyrexia and painful œdema of the subcutaneous cellular tissue is sometimes seen in children. In one case reported by MM. Barthez and Rilliet, a boy three years old was suddenly seized whilst in good health with pains in the feet, which soon became swollen. His skin was hot; on the third day there was general anasarca, and large round red patches appeared on the skin. Two days later, the skin of the legs became shining and tender. It was then covered with patches, some red and some yellowish, round, and varying from half a line to five lines in diameter. The red spots were very slightly elevated, surrounded in most cases by a pale rose-coloured areola, whilst the centre was of a claret colour, and did not disappear on pressure. The child was quite well at the end of a week.

Another case is described by Ollivier (Archives de Médecine, vol. xv. 1827, pp. 206–216). Here the disease was more severe, the ecchymoses being larger and more numerous: enteritis was also present. There were several successive crops of eruption, each one accompanied by the reappearance of œdema. These cases are considered by Barthez and Rilliet to be allied to Purpura urticans. No mention is made of the presence or absence of albuminuria in either of the cases. They seem to have been cases of erythema, complicated with extravasation into the cutis.

Causes.—These are not properly understood. Cutaneous hæmorrhage may arise under very diversified conditions, many of which have been very illogically assigned as causes. Impure air, indigestible or scanty food, and continued fatigue, by impairing the process of sanguification, are supposed to help on the development of the disease. Purpura frequently occurs as a sequela to small-pox, measles, scarlatina, and rheumatic fever. Intemperance appears to have induced it; damp lodgings and miasmatic influence favour the occurrence of hæmorrhage which has also occurred in the course of cirrhosis, acute atrophy, and cancer of the liver, of ague, of Bright's disease, of amyloid disease of the viscera, of syphilis, and of long-standing suppuration of bone. Aggravated jaundice is often attended with Purpura. In fact the old writers recognised some connexion between it and disease of the liver, in the use of the term *Purpura hepatica.* It has also been seen in persons not exposed to any specially debilitating causes, and apparently in good health. Amenorrhœa has been assigned as a cause; it is more probable that it was a common effect of some unknown cause. Purpura has been known to disappear after the occurrence of profuse menorrhagia. Ricord mentions the case of a syphilitic patient who suffered from Purpura hæmorrhagica, whenever he was treated with iodide of potassium. Virchow observed the same effect from the administration of this drug to a cancerous patient; and other writers have also recorded the occurrence of Purpura in patients when under the influence of the iodide of potassium. The want of vegetables in food, and an excessive quantity of salt meat, have been assigned as causes of Purpura; but this has arisen from confounding Purpura and scurvy. It may occur at any age, but is especially frequent in children and old people. There must be some condition common to these various diseased states leading to perhaps more or less softening of the tissues of the capillaries, and certainly rupture; but before referring to the exact nature of the changes in Purpura, it is necessary to say a few words on the character of those morbid conditions which have been found to accompany cutaneous hæmorrhages.

PATHOLOGICAL ANATOMY.—In the first place the petechias and ecchymoses in cutaneous hæmorrhages are due to extravasation of blood,—not merely of its colouring matter,—into the cutis. There is sometimes hæmorrhage into the subcutaneous cellular tissue, and into the muscles. Smaller or larger extravasations are also often found in the mucous membranes of the mouth, pharynx, stomach, small and large intestines. They are less frequently met with on serous surfaces, the pleura, pericardium, and peritoneum. Hæmorrhage in the lungs or in the cerebrum is not often seen. Blood is often found in the pelvis of the kidney. The blood in the body is in some cases quite natural, coagulating readily; in other cases it is unusually fluid, and indisposed to coagulate. The liver may be healthy, or the seat of incipient or advanced cirrhosis, of acute atrophy, or of amyloid degeneration. The spleen is either normal or enlarged. Dr. Habershon (Guy's Hospital Reports, Third Series, vol. iii., 1857) describes cases in which the spleen was large, of a dull-red colour, studded throughout with pale yellow spots from one to three lines in diameter, which were connected with the capillary circulation, and consisted of cells, nuclei, and granules. The white corpuscles of the blood were not in excess, but rather deficient in number. Dr. Ogle (Path. Soc. Trans. vol. xi. p. 269) describes cases of enlarged spleen containing adventitious material, of which white corpuscles formed a considerable proportion, and alludes to the circumstance that in several of them there was a tendency to purpuric hæmorrhage. The kidneys may be healthy, or the seat of chronic degeneration, either amyloid, or of some other character. The capillaries of the skin have been examined, and pronounced healthy; but this statement must be received with caution, owing to the difficulty of the examination. In the case of Purpura recorded by Dr. Wilson Fox,[1] occurring in a case of secondary syphilis, with severe ulceration of the pharynx and larynx, amyloid degeneration was detected in the spleen, kidneys, liver, and intestines; the same change was also observed in the muscles and the capillaries of the skin.

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[1] Brit. and Foreign Med.-Chir. Review, October 1865.

Dr. W. Fox states that "sections of the skin near, but not in the parts affected with hæmorrhagic extravasations, gave either with Schultze's solution (chloride of zinc and iodine), or with iodine alone, or iodine and sulphuric acid, a most intense reddish brown, in portions between the fat, besides corresponding to the course of the capillaries. This coloration did not pass much into the papillæ. The colour, with Schultze's reagent, was somewhat evanescent; but that with iodine lasted from forty-eight to seventy-two hours, and in some preparations the marking out of the capillaries was beautifully effected in this manner. This change was not constantly met with in all portions of the skin tested; but was best marked in portions taken in close proximity to the affected spots. In some of these parts, in which I succeeded in isolating portions of the capillaries and smaller arteries, I found that they broke up very easily, that some presented a peculiar glistening, waxy look, while others had a non-granular appearance, in no degree corresponding to the appearances observed in health."

In the same case, there was hæmorrhage into many of the muscles, and peculiar appearances, very similar to those described by Zenker, were seen in other parts of the muscle. "Patches of muscle, from a quarter to half an inch in diameter, are whitish grey, contrasting markedly with the normally-coloured tissue around. The fibres so affected resemble those of fishes. They are dry, friable, and break with a granular fracture. They have also a strong refraction. These portions of muscle stain of an intense reddish brown, with iodine."

"Under the microscope these pale patches presented a mixture of two appearances. (*a*) Some fibres were excessively pale, had a uniform waxy look, and had lost nearly all appearance of striation. Many broke up very easily, and in many places ruptured within the sarcolemma. Some presented an appearance as if made up of innumerable refracting particles, but I could not succeed in breaking up these fibres, so as to examine separately the constitution of these particular particles. These fibres were not distinctly enlarged. (*b*) Other fibres in the same field, much paler than natural, had not the glistening, waxy look, but were very finely granular. The transverse and longitudinal striation was indistinct; but these fibres, in many cases, split up very easily into fibrillæ. The granules disappeared for the most part under liquor potassæ or acetic acid, leaving a few scattered fat drops in the fibre. The nuclei appeared about as distinct as usual in the more waxy specimens; they were indistinct, but not enlarged in those which were granular.

"The exceedingly pale spots in the rectus abdominis presented the waxy appearance in the most marked degree. The heart showed, in a few parts, similar waxy fibres, but in the affected spots the more general appearance was that the fibres were finely granular, with indistinctness of the transverse striation. The granular character disappeared as a rule with acids and alkalies; a few fibres here and there were distinctly fatty."[1]

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[1] Wilson Fox, loc. cit.

An attempt was made to inject the vessels of the fore-arm after death; but although great force was used, the injection failed for the most part to reach the smaller vessels. The blood coagulated firmly; at an early stage of the disease, the white corpuscles of the blood were seen to be in excess, whilst at a later period they had decreased in number, and bore relatively to the red an apparently normal proportion.

Dr. Dickinson (Trans. Path. Soc. vol. xvii. p. 13) describes a case of meningeal apoplexy associated with Purpura. In a cachectic patient, sudden effusions of blood occurred in the skin of the arms and thighs, one being the size of an egg. Subsequently the man was seized with pain in the neck, rigors, vomiting, and want of sleep. He was restless and semi-delirious, but not paralytic. He could not speak, and death occurred the third day.

At the post-mortem examination the vessels of the pia mater were very carefully examined under the microscope, and they were found to be apparently healthy; the blood in the body was fluid, and like the juice of cooked cherries or plums. The liver was cirrhotic. No doubt bile products had been plentifully circulating in the blood. This case contrasts with that of Dr. Wilson Fox, in which the capillaries were diseased. The occurrence of Purpura in connexion with so-called amyloid deposit has, however, been noticed in other cases by Dr. Dickinson. The same gentleman has also (Trans. Path. Soc., vol. xiii. p. 104) recorded a case of Purpura in a girl aged 16, who was jaundiced from the pressure on the hepatic duct by an hydatid cyst, the blood containing a large amount of bile, but in which there was no appreciable lesion of tissue.

NATURE.—Much uncertainty exists as to the exact nature of the disease of which cutaneous hæmorrhage is the outward evidence. There must be rupture of the vessels to permit the escape of blood; and the latter does not take place in health be there ever so much pressure on the capillaries. When hæmorrhage does occur, it might—considering the matter *à priori*—be due to a weakened and diseased state of the capillaries, the result of changes in the blood it may be; or to some change in the blood which originates a softening of the tissues outside the vessels as well; or it might be due to a loss of nerve control over the vessels, so that the normal force of the heart's action could not be withstood. Now the blood has been several times analysed; it has sometimes been found to contain a deficiency of fibrin, sometimes a normal quantity, and sometimes an excess of that ingredient. Becquerel and Rodier have given the name of *scorbutic* condition to all cases in which the fibrin is less than 0·2 per cent. in the blood. In scurvy proper, however, and in Purpura, the fibrin may even be increased, so that a deficiency in fibrin is certainly not the property of the blood on which hæmorrhage always depends. Becquerel found in 1,000 parts of blood 803·44 water, and 196·56 solid matter.

In an examination of the blood in two cases of Purpura, by Dr. Parkes, the only remarkable result was an excess of iron with a general deficiency of the solid constituents. The relative proportion of the different organic solid constituents to each other was not much affected. In an analysis by Routier, quoted by Simon (Animal Chemistry, vol. i. p. 319), there was no general deficiency of solids, but a small proportion of fibrin, ·09 per 100. Simon examined the bloody fluid discharged from the mouth of a girl, aged twenty years, suffering from Purpura hæmorrhagica, and found it to contain no fibrin, a few blood-corpuscles, and some bile.

It was long a favourite doctrine, that change in the *quality* of the fibrin, rendering it liable to coagulate in a peculiarly gelatinous manner, existed in cases of Purpura and scurvy. Some modern observers (Magendie, Marchal, and Poggiale) have adopted this view, but it is one to which little importance can be attached; in the first place, because this form of coagulum is often absent, and in the second place, because nothing is known of the change which has taken place in fibrin which does thus coagulate. Deficiency of fibrin might help out the disease.

It has been proved by experiments on animals, conducted by Magendie, Virchow, and others, that the injection of putrid matter into the veins will cause ecchymoses of the endocardium, of the lungs, liver, and kidneys, &c., effusion of blood from the intestinal mucous membrane,—this may be by the production of thrombosis. The injection of ammonia, however, into the blood, has also been found to produce hæmorrhages. This shows that extravasations may follow changes in the blood, but proves nothing as to the nature of Purpura. Dr. Garrod has suggested that a deficiency of potash in the blood may be the cause of scurvy, and this disease has been cured by the administration of salts of potash; in this respect, Purpura is entirely different from scurvy. Purpura is not cured by the administration of salts of potash, or of fresh vegetables.

The occurrence of cutaneous hæmorrhage or Purpura in the more general sense of that term, in acute atrophy of the liver, as well as in cirrhosis and other diseases of that organ, has been variously explained by different writers. At one time it has been ascribed to debility of the heart's action, the general want of tone in the system, and the defective nutrition of the blood-vessels (Buhl); at another time, to the deficient formation of fibrin (Monneret); and at another, to an over-distended condition of the blood-vessels in consequence of a deficiency in the secretion of bile. Frerichs suggests that there is an abnormal attraction between the walls of the vessels, and the blood which has become altered in its composition, from which arise obstruction and rupture of the capillaries (Clin. Treatise on Diseases of the Liver, vol. i. p. 232, New Sydenham Society). The presence of a large quantity of bile in the blood has been stated to cause a solution of the walls of the blood discs, favouring the transudation of hæmatine into the tissues. It is questionable, however, whether change in the blood alone will account for the escape of blood corpuscles from the capillaries. If the blood be charged with bile, or morbid products, or be deficient in fibrin, and obstruction occur, then rupture might more readily take place. In Purpura there is probably always an actual escape of the blood discs; it is not merely an extravasation of the colouring matter, owing to a solution of the walls of the discs.

The sympathetic nervous system may possibly, besides the blood, be concerned in the recurrence of Purpura, for Simon produced cutaneous hæmorrhage by destroying several of the great sympathetic ganglia in the neck of the frog; softening of the tissues first occurring.

To explain satisfactorily an escape of blood-globules from the capillaries, one imagines that lesions of the vessels must be assumed to exist. A blood change may be the first morbid condition leading to these lesions. The nature of the changes in the capillaries is not yet ascertained; sometimes it may be simple rupture from overdistension; but more frequently there is probably a degenerative change in the capillaries, making them specially liable to give way as in the case of Purpura hæmorrhagica, above referred to, observed by Dr. Wilson Fox, where the capillaries were in process of lardaceous degeneration.

Dr. W. Fox observes: "Another very important question arises as to how far this affection of the capillaries can be logically considered to have been the cause of the hæmorrhage. Any direct association of the two changes will probably be considered doubtful by many who know that lardaceous affections of tissues are rarely associated with hæmorrhage; and further, that the change in the parenchyma of organs and in mucous membranes thus effected is often preceded by a similar change in the smaller vessels.[1] The evidence as it stands at present is decidedly against such a theory of causation, unless one or two possible hypotheses may be admitted to explain the connexion of the phenomena observed. (1) May this lardaceous degeneration, which we know chiefly as a chronic disease, occur occasionally in a more acute form, and in this manner so rapidly alter the elasticity of the vessels, before their diminished calibre can have retarded the flow of blood in the part, that rupture and hæmorrhage ensue? (2) Is it possible that this lardaceous change, occurring only in *tracts* of tissue, may throw such a stress on the collateral capillary circulation of the tissue around, that adjacent but comparatively unaffected capillaries gave way? Both theories derive some support from the observations made on the dissemination of the degeneration in this case, and also from the observations of Zenker, in a similar degeneration of the muscles in typhoid fever."[2] But then we have the testimony of such cases as those of Dr. Dickinson to the contrary, in which the capillaries seemed to be quite healthy.

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[1] Virchow, Cellular Pathology. Chance's Translation, p. 374.

[2] Ueber die Veränderungen der willkührlichen Muskeln in Typhus Abdominalis. Leipzig, 1864.

Dr. W. Fox's case of Purpura, though not an uncomplicated one, seeing that the patient was suffering also from an aggravated form of secondary syphilis, must be regarded as a valuable contribution to the pathology of Purpura; it may serve as an indication of the direction in which inquiries as to the nature of the disease should be instituted.

Purpura senilis is probably due to a degeneration of the capillaries, in parts exposed to rough usage; we are not aware that this has been confirmed by actual observation.

The general result at which we arrive as regards cutaneous hæmorrhage then is this; that it occurs in many diseases in which the blood is disordered. When—this is Frerichs' view—in consequence of the altered relation between the blood and the tissues, the former passes with difficulty through the capillaries, and the tissues are more or less badly nourished in common with the whole body, rupture of the capillaries is likely to occur, and especially if there be, in addition, amyloid degeneration of the capillaries or obstruction to the general circulation, as in liver disease. This applies to cutaneous hæmorrhages as a whole. In the disease more properly called Purpura, there is no evidence of diseased capillaries, nor of blood-change. Hebra, however, asserts that the capillaries must be weak, and that Purpura is "due to change in the cohesion of the tissues surrounding the ruptured capillaries," as in inflammations. Hebra thinks that temperature and clothing have much to do with the occurrence of Purpura. It still remains a question how far in progress of time a common pathological origin will be assigned to all cutaneous hæmorrhages, Purpura included.

PROGNOSIS.—In Purpura simplex recovery usually occurs, but relapses are very frequent. Purpura hæmorrhagica is always a grave disease; it has often proved fatal from exhaustion, or, more rarely, from cerebral or pulmonary apoplexy. Recovery ensues in a certain proportion of cases under judicious treatment. The prognosis of cutaneous hæmorrhage is that of the general disease in which it occurs.

DIAGNOSIS.—The characteristic features of Purpura are the extravasation of blood in the cutis, and, in the hæmorrhagic variety, loss of blood from other parts. The eruption in the skin is known from other non-hæmorrhagic eruptions, by its not disappearing or fading on pressure. *Typhus fever* and *hæmorrhagic measles* will be distinguished by the constitutional symptoms and history peculiar to those diseases. In the former a history of contagion, destitution, or overcrowding, cerebral symptoms, dryness of the tongue, and the fact that the spots are small, generally spread over the body, and at first disappearing on pressure, will prevent any mistake; in typhus, too, there are no hæmorrhages from mucous surfaces, unless it be complicated with scurvy. In measles, the rash is brighter, and at first disappears on pressure; there are also signs of coryza and bronchial congestion, with pyrexia, preceding the rash for forty-eight hours or more. —The only disease with which Purpura is often confounded is *scurvy.* The latter disease is always caused by a want of fresh vegetables; it may be always prevented, and generally cured by a proper supply of vegetables. This is not true of Purpura. In scurvy, the gums are usually swollen, soft, and sore, which is not the case in Purpura. There is a tendency to painful swellings of the limbs, and stiffness of the joints, in scurvy, not observed in Purpura. The gums are swollen, spongy, discoloured and bleeding in scurvy. The eruption of Purpura is often bright at the onset, and the lips and tongue are frequently of a good colour when the disease sets in. In scurvy, the complexion is pale and sallow, and the patient is obviously out of health before the hæmorrhages take place; this is very often not the case in Purpura. Fresh vegetable diet at once prevents the occurrence of hæmorrhagic spots, and does not influence Purpura.

Ecchymoses from violence may be known by their fixed local character, their size, and the absence of small petechiæ. Flea-bites in unhealthy persons often retain the petechial character for many days, or even weeks; they may be known by their nearly uniform size, and the presence of a central minute point where the skin was punctured by the insect.

The *hæmorrhagic diathesis,* in which there is a tendency to excessive loss of blood from very slight causes, must be distinguished from Purpura.

In persons of a hæmorrhagic diathesis, there is usually some wound, ulcer, or contusion, of the part from which blood escapes; there is not the tendency to general bleeding in all parts of the skin and mucous membranes which characterises Purpura. Signs of the diathesis generally make their appearance at an early age, and there is very often a history in the family of an hereditary tendency to the same condition.

Treatment.—According to the different views entertained of the nature of Purpura, different lines of treatment have been recommended and adopted. The cases brought together under the name of Purpura, in its wider signification, being of very varying nature, agreeing only in the circumstance of presenting hæmorrhage in the cutis, obviously must not be all treated alike. Regard must be paid to the diathesis of the patient, whether he be rheumatic, gouty, cancerous, or scrofulous. The antecedent conditions must be ascertained—whether his diet has been suitable or deficient in any nutritive elements; whether he have resided in damp, ill-ventilated places, or been exposed to malaria.

At one time, blood-letting was recommended for Purpura, as for nearly every other disease. Its use is now universally discarded. If there be signs of plethora, with a full, strong pulse, and the mucous membranes of a good colour, the use of free saline purgatives, preceded by a dose of calomel, is indicated. The purgative may in rare cases be combined with a small quantity of antimony. Cases of this kind are seldom met with in the present day.

If there be any sign of hepatic congestion, the sulphate of magnesia or soda,[1] with dilute sulphuric acid, should be given two or three times a day; a good dose of jalap, with a grain or two of calomel, may be first given. The perchloride of iron has been much extolled in France and elsewhere. M. Pize (Journ. de Méd. et Chir. pratique, August 1860) regards it as an almost infallible cure administered in quantities of one drachm in twenty-four hours (equal to about four drachms of the tincture of the sesquichloride of iron of the London Pharmacopœia). If iron be given at all, it should be used in full and frequently repeated doses, half a drachm or a drachm of the tincture of steel every two hours. In the ordinary cases of Purpura simplex, the dilute sulphuric acid, with quinine and sulphate of magnesia, may be given.

Dr. Neligan has strongly advocated the use of turpentine in large doses, in Purpura, with or without castor-oil. He gave from an ounce to an ounce and a half to adults, and from two to four drachms to children. He reports several cases in which it certainly seems to have exerted a most remarkably beneficial influence.

I have myself administered it with very satisfactory results to two children affected with this disease, one in a very severe form. I have seen it used in two fatal cases in which it appeared to check the hæmorrhage, but its use was resorted to at so advanced a period of the disease as not to afford any reasonable hope of preventing death.

In one child, aged 11 years, who had numerous petechiæ and ecchymoses of the skin, with profuse epistaxis, and was losing blood from the kidneys, the bowels, and the stomach, I gave at first two drachms of oil of turpentine, with half an ounce of castor-oil, once a day; on this treatment there was slight improvement. She was, at the end of two days, ordered olei terebinthinæ, ʒj.; mucilaginis, ʒj.; syrup ad ℥ss.; ol. menthæ pip. ♏ij. twice a day; and an enema consisting of olei ricini, ℥ss.; olei terebinthinæ, ℥ss.; decocti hordei, Oss. once a day.

In four days a marked improvement had taken place; all hæmorrhage had ceased, and the ecchymoses were rapidly disappearing. The turpentine was then given once a day for about a week, at the end of which she was quite well, regaining her colour and strength.

Dr. Hardy, of Dublin (Dubl. Hosp. Gazette, 1859), advocates the use of the tincture of larch bark, in doses of about ten or fifteen drops every hour, or less frequently, according to the severity of the case. This remedy, probably, acts in the same way as turpentine; it is said to be an agreeable medicine to take.

When the hæmorrhage is very profuse and the patient is exhausted, astringents, such as gallic acid and acetate of lead, are usually resorted to, but they are very often inefficacious.

Mr. Hunt recommends arsenic in Purpura simplex, and in this recommendation he is supported by Dr. Habershon, upon what ground it is difficult to say.

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[1] This salt has been strongly recommended in persons of the hæmorrhagic diathesis.

In treating a case of Purpura, regard should always be had to the circumstances of the patient prior to the attack. If the diet have been deficient in quantity or quality, such deficiency should be made up by a well-regulated diet, a due proportion of animal and vegetable food being administered, with a moderate allowance of light stimulant, such as claret, marsala, or some pure wine. If the pulse be full and firm, and the colour of the lips good, an emetic of ipecacuanha, or even antimony, at the outset may be a good introduction to the turpentine treatment. Digitalis has been recommended in cases where the pulse is very frequent, but not strong enough to justify blood-letting. This drug has been found useful in menorrhagia and in pulmonary hæmorrhage ; it may be worth a further trial in Purpura hæmorrhagica.

Local measures may be resorted to where there is great loss of blood from parts within reach. For epistaxis and uterine hæmorrhage, injection of cold lotions containing alum, tannin, or acetate of lead, may be used, or plugging may be adopted in extreme cases. In hæmatemesis, ice may be given by the mouth. Purpura of the extremities may often be checked by careful bandaging of the part.

In Purpura simplex, following rheumatism, quinine is often of use.

Hebra advises in all cases that the patient should take *rest*, and it is always desirable to keep the patient, if possible, in the recumbent posture for some few days.

In convalescence, the administration of bark, as recommended by Werlhoff, Behrend, and Jeller, who first described the disease, may be had recourse to, if there is any symptom of debility.

RICKETS.

By W. Aitken, M.D.

Definition.—A constitutional disease, characterised by an unhealthy state of the system, which precedes for several weeks or months a peculiar lesion of the bones, and of some of the solid visceral organs. The lesion in the bones is characterised by irregularity in their growth, by non-solidification of the growing layers of the bone, by the progressive formation of medullary cavities in the older or more mature bone, thus rendering the bony laminæ thin and brittle (Virchow). There is generally albuminoid (amyloid?) degeneration of the spleen and liver.

Synonyms.—Morbus Puerili Anglorum, the Ricketts;[1] Flagellum Angliæ seu Tabes Angliæ; Rhachitis;[2] Rachitis;[3] Atrophium infantum Anglicum;[4] Cyrtosis Rachia;[5] Cyrtonosus; Tabes Pituitosa; Morbus Anglicus; Osteomalachia infantum;[6] Tabes Pectorea; Spina Nodosa; Rachitismus; Osteosarcosis; Innutrio Ossium;[7] Osteomalakia;[8] Scrofula Rachitis;[9] Rachite, Rachitisme, Riquets, Noruire; Englische Krankheit, Zweiwuches; Rachitide.

Causes.—As with other constitutional diseases, so with Rickets, the transmissibility of a defective constitution from parent to offspring is the first link in the chain of causation which brings about the cachexia or unhealthy state of the system peculiar to Rickets. Wiltshire, from personal experience, is of this belief. Herring affirms true Rickets to be, in the highest degree, hereditary; while, according to Schönlein, too early marriages, and, according to Küttner, intermarriages, mainly conduce to its transmission. Sir W. Jenner, on the other hand, knows of no facts to prove that Rickets is hereditary.

The history of the disease, however, appears to show that there are *predisposing causes* derived from the parents or the nurse, which are so capable of influencing the health of the child, as to lead in course of time to the establishment of the disease now under consideration. This is quite different from a child inheriting Rickets as a disease which its parents had, and which they transmitted to its constitution.

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[1] Whistler.
[2] Glisson.
[3] Sauvages, Vogel, Boerhaave, Cullen.
[4] Chuden.
[5] Good.
[6] Cumin.
[7] Darwin.
[8] Swediaur.
[9] Young.

Of predisposing causes derived from the parents, the influence upon children begotten of bodies exhausted from chronic disease, venereal excesses, or age, has been recognised from very early times. An innate or congenital debility is thus imparted to the infant constitution; and when Rickets becomes a frequent disease (as, at one period, it seems to have been in this country), its occurrence would often seem to be hereditary. Vogel says, the father of a rickety child has acknowledged to him that his system was contaminated by syphilis at the time the child was begotten; and Vogel is also of opinion that the existence of constitutional syphilis in the parent may frequently explain the occurrence of Rickets in the children of the better classes. Moreover, it is an ancient belief which regards Rickets as a degenerate form of syphilis. Nevertheless, Sir W. Jenner is opposed to the belief that Rickets is a form of congenital syphilis, mainly by a consideration of the following facts :—" The parent who infects his offspring [with syphilis] has usually contracted syphilis before marriage, and the children first begotten after infection are those who suffer [from inherited syphilis]; while, as a rule, it is only the younger children of a family that suffer from Rickets—the first born being commonly healthy, though the later born are highly rickety." Sir W. Jenner is also very sceptical as to the influence of the father in affecting the health of the children; but he believes that the health of the mother has a decided influence in the development of Rickets in the child. Of this he is sure, that where the mother is of delicate health—in a state in which anæmia and general want of power form the prominent features, without being the subject of actual disease—that such mothers give birth to children which are often apt to be affected with Rickets to a most decided degree, even although the father is in robust health, and when the hygienic conditions under which the children are placed may be most favourable.

With reference to the development of Rickets after birth, the experience of Sir W. Jenner further leads him to state that it is very common for the first, or the first two or three children, to be free from any signs of Rickets, and yet for every subsequent child to be rickety; and, if a woman bear one rickety child, in the majority of cases all her subsequent offspring will be rickety. This he explains upon the following grounds : " That, among the poor, the parents are generally worse fed, worse clothed, and worse lodged, the larger the number of their children—for the man's wages remaining stationary, the calls on his means are increased. And among the rich and the poor alike, the larger the number of children, the more has the mother's constitutional strength been taxed, and the more likely is she to have lost in general power." (Medical Times and Gazette, March 1860.)

The causes of the peculiar ill-health which precedes the expression of Rickets in the child, are the subject of great differences of opinion; but generally it may be stated that whatever external or extrinsic circumstances are favourable to the formation of watery blood (hydræmia) in a child, seem favourable to the development of Rickets. Deficient or improper diet taken daily, impure air constantly breathed, deficient exposure to solar light, want of cleanliness, cold, moisture, want of exercise in the open air, are all, more or less, influential in the production alike of Rickets, and of other constitutional diseases. But details, relative to the specific kind of improper food, although of most interest, are the least known. The influence of improper food has been fully proved by the experiments of Guérin on animals. On the other hand, Dr. W. Cumin, in his article on Rickets in the Cyclopædia of Practical Medicine, observes, that whole broods of young geese and ducks, young pointer and greyhound puppies, and young pigs, have Rickets or the "krinckets," when they have been continually exposed to cold and wet, or have been kept in damp kennels and sties. Again, M. Trousseau states that of a hundred rickety children, ninety-eight were either never suckled at all, or were weaned very early; while the experience of Sir W. Jenner and of Mr. Lonsdale confirms the belief that the improper and unsuitable feeding of children is by far the most efficient cause of Rickets, the nourishment being first of all deficient during the period of infancy, when the child does not get sufficient from the mother, either in the quantity or in the quality of the milk secreted; and Mr. Lonsdale has invariably found, that in all rickety children, the parents have had little or no milk for their supply, and have been obliged to feed the children either partially or wholly with food other than the milk of the mother. The mothers observe that the children never grew properly from the first, and it is mainly the *improper* nature of the supplementary food, "given by hand," which impairs the health of the child. Here the evidence of Sir W. Jenner corroborates and completes the evidence of Mr. Lonsdale. Among the poor, Sir W. Jenner observes that the children are dosed with *improper* food even from their birth; and the common mode of rearing the children of the poor in London is thus described by him:—"For the first two or three days after birth, their tender stomachs are deranged by brown sugar and butter, castor-oil and dill-water, gruel and starch water; as soon as the mothers' milk flows, they are, when awake, kept constantly at the breast. And well for them if they are not again and again castor-oiled and dill-watered, and even treated with mercurials,—for the poor have learned the omnipotent virtues of grey-powder.

"After the first month, bread and water sweetened with brown sugar is given several times a day, and during the night the child is, when not too soundly asleep, constantly at the breast. As soon as the little ill-used creature can sit erect on its mother's arm, it has at parents' meal-times 'a little of what we have'—meat, potatoes, red herring, fried liver, bacon, pork, and even cheese and beer daily, and cakes, raw fruits, and trash of the most unwholesome quality, as special treats, or as provocatives to eat, when its stomach rejects its ordinary diet. Then, instead of being weaned when from ten to twelve months old, the child is kept at the breast when the milk is worse than useless, to the injury of the mother's health, and to the damage of its after-brothers and sisters, in the hopes that thus keeping it at the breast may retard the next pregnancy. The children are sacrificed that the passions of the parents may not be restrained." (Med. Times and Gazette, May 12, 1860.)

Nevertheless, the causes of Rickets are still extremely obscure, as are the causes of all the constitutional diseases. The extensive exciting causes referred to are common antecedents of many affections, and seem to induce one or other of these diseases, according to circumstances of which, as yet, we know nothing.

The subject requires and merits extensive investigation, for, primarily or secondarily, Rickets causes more deaths than any other disease of childhood; and, looking to what we do know of the unhygienic circumstances under which it has been developed, it is one amongst the most preventible of diseases. Without doubt, Rickets is "the most common, the most important, and, in its effects, the most fatal of diseases which exclusively affect children." (Jenner.)

SYMPTOMS.—The commencement of Rickets is said to have been recognised in the fœtus. Bordenave and Pinel have mentioned such cases. The latter describes the case of a rickety fœtus at the eighth month, in which the distortion of the bones was confined chiefly to the lower limbs. Meyer also more recently records a case of *intra-uterine* rachitis. (Henle and Pfeuffer, Band. vi. § 151, quoted by Wiltshire.) Glisson, Henckel, Klein, Lelletier, and other authors referred to by Dr. Cumin, have also given examples of congenital Rickets.

Sir W. Jenner, however, in his extensive experience has never seen congenital Rickets, but he has often heard the mother say, that the rickety deformity of her child had existed from its birth; although he properly attaches no value to such assertions if unsupported by strong confirmatory evidence.

The earliest recognition of the cachexia associated with Rickets has been variously stated, and these statements have no doubt varied with the opportunities of the observers for seeing cases at an early period of the disease. Opinions, therefore, on this point are somewhat conflicting.

According to Jenner, the impairment of the general health (cachexia) rarely becomes apparent before the fourth month, and usually between the fourth and twelfth month. (Med. Times and Gazette, April 28, 1860.)

Rickets, however, is rarely observed before the seventh month (Cumin); "and most commonly does not declare itself until the child first begins his attempts to walk, or until he suffers from the effects of the first dentition." (Cyclop. of Pract. Med. vol. iii. p. 616.)

Dr. Mason Good records, that "Rickets seldom appears earlier than the ninth month of infancy, and not often later than the second year." (Study of Medicine, vol. v. p. 346.)

Dr. Copland, who has had considerable experience of the disease, is still less definite. He states that "the complaint has been met with from the earliest months, till approaching puberty; but it is most commonly observed to commence during the first dentition, or from six or seven months to three years of age." (Med. Dict. vol. iii. p. 643.)

Dr. Dunglison believes that children are unquestionably born with a *predisposition* to Rickets, although they rarely exhibit any evidence of the disease till towards the termination of the first year; and at first the progress of the disease is so very slow as almost to be imperceptible. (Practice of Medicine, vol. ii. p. 704.)

All these observers agree, however, that the complaint is one peculiar to infancy; and, as such, it is materially associated with the development of the child—perhaps with the conception of the embryo—at all events, its phenomena are *inbred,* and undoubtedly, therefore, constitutional. Some few cases occur of children born with disproportionate development of the bones, which have led to the belief that Rickets has been observed in the fœtus; but, as Dr. Copland justly observes, "It is doubtful whether or not the imperfect ossification, and consequently softened state of the bones observed congenitally, should be viewed as Rickets; as, in this disease, there is a change in the state of the affected bones, different from a mere delay or simple imperfection of osseous formation." (Loc. cit.)

Out of 346 cases of Rickets observed by M. Guérin, there had origin:—

Before birth	3 cases.
In the First year	98 ,,
,, Second year	176 ,,
,, Third year	35 ,,
,, Fourth year	19 ,,
,, Fifth year	10 ,,
,, Sixth year	5 ,,
	346

Of these, 148 were males, and 198 were females.

From these records, the number of cases happening in the first and second years of life very greatly exceeds those of other periods of life; and in the subsequent years the numbers diminish very rapidly.

It seems to take a considerable length of time before the phenomena characteristic of Rickets become so fully expressed as to leave no room for doubt regarding the nature of the affection. According to Guérin, there is a period of at least six months during which a marked series of deranged actions succeed each other. But while many of these are common to other diseases, there are a few characteristic phenomena which, while they occur in sequence, are so peculiar as to be sufficient to determine the specific characters of the disease now under consideration.

The phenomena and symptoms of Rickets, therefore, may be arranged under four classes, namely :—

First, those which are common to many diseases, and in which there is nothing diagnostic of Rickets—symptoms which might arise from deranged digestion, from improper food, or from tuberculosis; and, as Sir W. Jenner observes, they are symptoms too often referred to "the irritation of teething," or to the so-called "infantile remittent fever." This has sometimes been described as the precursory or incubative stage. (Guérin, Guersent, Copland.)

Second, those which "at once mark the nature of the disease, render the diagnosis easy, and enable us to predicate that the true affection will show itself."

Third, the stage of characteristic deformity.

The *fourth* class of phenomena may be of favourable or unfavourable import; and may characterise a period of restoration to health, of irremediable atrophy, or of approaching dissolution. The symptoms peculiar to each of these stages are described from the writings of physicians who have had extensive experience and opportunities of observing this remarkable disease—namely, MM. Guérin and Guersent in France, and Copland and Jenner in this country; the latter of whom has given, from his own observation, the most detailed and original account of this disease that has ever been given by any physician. That account will be found in the pages of the Medical Times and Gazette, vol. i. for 1860.

During the precursory or incubative period, the most ordinary symptoms of impaired general health are those which indicate gastro-intestinal irritation. The bowels are irregular in their action, sometimes confined, or only relaxed, but more commonly there is diarrhœa, with tumidity or enlargement of the abdomen, when emaciation generally becomes more or less obvious.

The stools are usually unhealthy-looking, pale, deficient in bile, or of a dirty-brown or leaden colour, and of a most offensive odour. In some respects this odour is peculiar in its resemblance to rotten, half-decayed meat. Appetite is feeble, capricious, or entirely lost; and digestion is difficult. The child becomes dull and languid, sad, or peevish, and averse to play or to exert itself in any way. There is low, febrile irritation present; the skin is hot; the temper irritable; and, although drowsy, the child sleeps but little. It is thirsty, and will drink large quantities of water; if it has begun to walk, it "is taken off its legs;" it lies about, and is unwilling to play or to be amused, or to indulge in any kind of action, preferring to sit or lie, and appearing to be feeble or indolent, and unable to use exertion of any kind. It complains of pains in the joints or along the bones; the face becomes pale, and the flesh becomes soft and flabby; the pulse becomes quick, and indicates irritability of the heart's action; the superficial veins become large, and the jugular veins especially are much dilated; the hair continues thin upon the scalp, and the fontanelle remains widely open.

But in each or all of these phenomena there is, as yet, nothing diagnostic of Rickets. They may accompany or usher in other maladies. The transition from apparent health to the expression of these phenomena is always gradual, more or less slow, and as Dr. Copland has observed, some other disorder or malady may mark the incubative stage of Rickets, and, overlaying as it were that disease, may render the possibility of diagnosis still more protracted and difficult. He refers especially to bronchitis, hooping-cough, and lobular pneumonia, and thus, instead of beginning abruptly, Rickets may commence and progress most insidiously, so that the mother cannot say when her child first began to suffer, and often the deformity and change in the shape of the bones are the earliest abnormities she observes.

On the other hand, Sir W. Jenner, from very extensive experience, has at last been able to identify three very distinct symptoms as commonly present during the approaching development of Rickets, which at once mark the nature of the disease and which render the diagnosis easy, and enable us to predicate that the bone affection will show itself.

These symptoms ought, therefore, to stand as they here stand—by themselves in the *second* class of phenomena. The most remarkable of these symptoms are as follow :—

(*a*) *Profuse perspiration of the head ; or of the head, the neck, and upper part of the chest.* Not uncommonly, says Sir W. Jenner, "it is because this symptom has arrested the mother's attention that she seeks medical aid. She uses the strongest terms to express the amount of the perspiration : 'It stands in large drops on his forehead !' 'It runs in streams down his face !' 'His head is all of a reek !' 'The pillow is soaked !' It is especially when the child sleeps that these copious perspirations of the head occur, but they are not infrequent at other times, as when the child is at the breast, or even resting its head on the mother's arm. A little increased exertion or a little increased temperature may induce them at any time." (Med. Times and Gazette, April 28, 1860.) Such perspirations are extremely weakening and colliquative during sleep, and when they occur the superficial veins of the scalp are generally large and full, the jugular veins much dilated, and sometimes the carotid arteries may be felt strongly pulsating. (Copland, Jenner.) While the skin generally may be moist during the day, during the night the abdomen and extremities are usually dry and hot, at the time when the head, face, and neck are bathed in perspiration.

(*b*) The second symptom recorded by Sir W. Jenner as specially indicating the general derangement which is the precursor of the rickety deformity of the bones, is *the desire and the efforts made by the little patients to be cool, particularly at night.* The child kicks the bed-clothes off, or throws its naked legs on to the counterpane. "He is always catching cold, because he will lie without any clothes at night," is the constant language of the mother in such cases. Sir W. Jenner says he has often gone into the wards of the Hospital for Sick Children, after the children have been some time asleep, and seen the rickety ones lying exposed; he has been assured by the nurses that they had put the bed-clothes over them again and again, but to little purpose; and this even in cold weather, when the other children were well covered. "During this restlessness at night the little patient may be seen frequently to bore its head into the pillow, or to rub the back of the head on the place where it rests. When it wakes in the morning, it will cry if the occiput is pressed, or when the head is lifted up and washed. If the head is examined, it will generally be found more or less denuded of hair. The whole skull feels thinner than usual, as if distended like a bladder, or it is flattened behind, at the vertex, and protruberant in front." The cranial bones may now be found "very soft in certain points, yielding elastically, like card-board, beneath pressure, and giving the feeling as if the skull might be bent inwards by the finger-points upon the brain." (Wiltshire.) This rachitic softening of the cranium has been now shown to be extremely common; and, in extreme Rickets, is generally the first manifestation of the disease, so far as the bones are concerned. (Elsäser, Widtman, Wiltshire.)

(*c*) The third characteristic symptom referred to by Sir W. Jenner is *general tenderness.* "The child cannot be moved without its uttering a cry; pressure on any part of its body is followed by like evidence of suffering." "He is tender all over," says the mother; or, "I can't think what has come to the child; if I do but touch him, he cries." The condition contrasts most forcibly with that of a child in health. "A child in health," says Sir W. Jenner, "delights in movements of every kind. It joys to exercise every muscle. Strip a child of a few months old, and see how it throws its limbs in every direction; it will raise its head from the place on which it lies, coil itself round, and grasping a foot with both hands thrust it into its mouth as far as possible, as though the great object of its existence at that moment was to turn itself inside out. The child, suffering severely from the general cachexia which precedes and accompanies the progressive stages of the bone-disease in Rickets, ceases its gambols, it lies with outstretched limbs as quietly as possible, for voluntary movements produce pain." But not only is the child unwilling to be moved, it is often in terror lest it may be moved, and it will cry at the approach of those who have been accustomed to dance it, and whose presence was wont to afford the child extreme pleasure. (Stiebel, Jenner.)

Dr. Copland, however, would be still more convinced if a series of symptoms which may be regarded as a fourth set of pathognomic signs of Rickets, were superadded to the three characteristic sets of phenomena just described.

When with the phenomena already mentioned, the urine is more abundant than in health, and when it deposits a copious calcareous sediment, or abounds in the phosphates, then the early or precursory stage of the complaint may be considered as already present; and it is in this stage especially that the salts are most abundant in the urine.

As the disease progresses, the general appearance of the child becomes more and more significant of the disease. It acquires a peculiarly staid, steady, or sedate aspect. Its natural lively expression is replaced by one of languor, of pensiveness, and of age. Its face grows broad and square; and when the child is placed upright on its mother's arm, it sits (as she says) "all of a heap." Its spine bends, and its muscles are too weak to keep it erect. The head thus comes to sink between the shoulders, and the face turns a little upwards. These symptoms, which Sir W. Jenner has so carefully and minutely detailed, as a painting from the life, are so very rarely correctly interpreted, and their significance so little appreciated, that he has subjoined the following case in illustration of the characteristics of this remarkable disease:—

"A. V., aged 3½, a male. His present ailment commenced about four months since, shortly after 'a severe cold on the chest,' with the following symptoms:—heat of skin, especially at night; thirst; loss of appetite; profuse sweating about the head; extreme tenderness of the whole body, so that he could not be touched without crying from the pain it caused him; relaxed bowels, the stools being, to use the mother's own words, 'stinking,' a 'rotteny smell;' a desire to lay exposed at night—again to use the mother's words—'even in that bitter cold weather he would never lay covered over; in the previous winter he liked to lay warm.' Although he had long run alone, he was soon 'taken off his legs.'"

Present State.—"Rather thin; muscles very flabby; evident tenderness of head, trunk, and extremities. The muscles seem to partake of the tenderness, and the abdominal muscles are as tender as those of the thighs. Sits in his chair, unwilling to move from morning to night. Cries if his brothers or sisters approach him. Feverish at night; throws his clothes off; sweats over the head profusely; the perspiration is limited to the head; appetite very small; bowels act once a day, but stools very offensive. Intellect decidedly less acute than that of his brothers and sisters was at the same age. Head large, square. He cut all his teeth long before his illness commenced. Spine curved backwards from about the first dorsal vertebra to the sacrum, and forward from first to last cervical vertebræ. Ribs very soft, so that there is great recession of each rib where it joins the costal cartilage at each inspiration. Physical signs of trifling catarrh. Very little enlargement of the ends of the long bones. No enlargement of glands, liver, or spleen." (Med. Times and Gazette, April 28, 1860, p. 416.)

The next set of phenomena which challenge attention in cases of Rickets are those which are associated with the characteristic deformity of the skeleton, when the consequences of the bone-disease are superadded to the general derangement of the system. If the general ill-health be severe enough to attract the notice of parents, it will not seem to have lasted long before the deformities of the bones begin to show themselves; and if the general ill-health be very much expressed, the softening of the bones will be so great as to render the deformities more remarkable than the mere enlargement of the ends of the long bones—usually so characteristic an appearance; and the younger the child the softer usually are the bones. With increasing pallor of the skin and flabbiness of the muscles, the extremities of the long bones (such as those of the ankles and wrists, and the sternal ends of the ribs) indicate, by a swollen and knobby, or double-jointed appearance, that the lesions of the skeleton are advancing so as to cause the deformities characteristic of Rickets. The lowermost ends of the long bones then begin to yield, especially those of the lower limbs; and hence, if the child has been able to walk, it can no longer do so. The bones gradually change their form. Those of the lower extremity become bent, so that the convexity of the curvature is forwards and outwards, and, of course, the concavity inwards and backwards. The femora are sometimes curved forwards, so that the convexity is forwards; and this forward curvature is produced before the child walks, simply by the weight of the legs and feet—the lower portion of the limbs hanging pendant from the knee-joint, as the child sits in its mother's lap or on a chair. The soft femur then yields, so that it is curved forwards. After the child walks, the weight of its body mainly determines the curvature of the thigh-bones; the curvature is then apt to be mainly outwards, and the curve which existed before walking will be exaggerated. In some cases the heads of the femora will be bent at an obtuse or acute angle to the shaft. More often, therefore, like the tibiæ, they are curved, so that the convexity is forwards and outwards. When the tibiæ curve before the child walks, it is an exaggeration only of the normal curve of the tibiæ in the young child, and is produced as the child sits cross-legged, leaning on to the floor or bed with the outer malleolus. The bones which, in health, seem to be the strongest, may be bent by the most trifling force; and if this force is constantly applied, so will the bending and deformity become more and more apparent, and the direction of the curves will depend on circumstances which determine the point on which the chief amount of pressure is brought to bear.

If the disease becomes developed during the later periods of childhood, the knees are then generally bent inwards, and the feet thrown outwards. The knees thus press against each other, and the child rests on the inner aspect of the foot; but the continued curvature outwards of both femora and tibiæ, may cause the knees to be separated to an unnatural distance. The whole of the lower limbs then form irregular curvatures, with the convexities outwards, and generally greatest at or near the knees. (Copland.)

The spine is so bent that the cervical anterior curve is increased. The face is thus directed upwards, and the head falls backwards; and this deformity becomes the more strongly marked according as the muscular debility is the greater. When this muscular debility is extreme, the head is no longer supported. It therefore falls forwards or backwards as circumstances may determine—waggles about, in fact, not unlike a button loosely attached to a garment.

There are two characteristic curvatures of the spine to be distinguished, according as the child is able or unable to walk. If the child is unable to walk, there is a posterior curvature of the spine, commencing at the first dorsal, and extending to the last lumbar vertebra. If, on the contrary, the child is able to walk, then this posterior curvature is limited to the dorsal region, but is combined with an anterior curvature in the lumbar region. This posterior curvature in the child yet in arms, is sometimes so extreme, that Sir W. Jenner has known it to be mistaken for angular curvature; and, as it may be so easily mistaken, he gives the following details as to how the curvature of Rickets may be distinguished from angular curvature :—

"If a child be held by the upper part of its trunk, the weight of the lower limbs will usually remove the rickety curve, and it may certainly be straightened, if the nurse hold the child by the upper part of the trunk, and the physician raises the lower limb with one hand, and at the same time places the other on the curved spine." (Medical Times, March 17th, 1860.)

These curvatures in Rickets are merely exaggerations of the natural curve of the spine, which always more or less exists when a child of three or four months old sits unsupported on the nurse's arm. Lateral curvatures, in the young child, are thus less common than those which are antero-posterior. Their direction in Rickets, however, is mainly determined by the position which may be accidentally assumed by the child; and, as Sir W. Jenner points out, if the child be carried constantly on the left arm, there is a disposition to lateral curvature, and the convexity of the curve will be towards the left. Dr. Copland gives the result of his experience in favour of the curve of the spine being in general outwards; but it is sometimes also lateral, outwards in the back or between the shoulders, where the curvature is also to one side, and to the opposite side in the lumbar region, where also there is sometimes a curvature inwards.

The curvatures of the spine, especially those outwards, are generally associated with a flattening of the ribs laterally. (Dict. of Medicine, vol. iii. p. 644.) The deformity entailed upon the thorax thus comes to be that which is of the greatest interest to the physician, because it is the one which is associated with the greatest distress and impediment to the functions of the heart and lungs. The back is flattened, the ribs being bent at an acute angle where the dorsal and lateral regions unite. Beneath each axilla there is a large concavity or hollow, instead of the normal rounded form. At this part the lateral diameter of the thorax is the greatest, and the ribs pass forwards and inwards from their angles to the points where they unite with their cartilages, so that on the line of junction of the ribs with their cartilages the lateral diameter of the thorax is the least; the cartilages curving outwards before turning in to unite themselves with the sternum. The sternum is thus thrown forwards, and the antero-posterior diameter of the thorax comes to be abnormally great; the sides of the chest are compressed, the dorsal spine pushed outwards (backwards?), and the sternum outwards (forwards?); and the diameter of the chest from left to right being thus diminished, while the antero-posterior diameter is increased, the deformity known as "pigeon breast" is formed. (Jenner, Copland.) The general aspect of the thorax is also otherwise changed, so that grooves are formed on each side of the sternum, where the ribs and cartilages unite. These grooves (as Sir W. Jenner describes them) pass from above downwards, on the antero-lateral aspect of the chest, and extend from the first to the ninth or tenth rib; and the deepest part of the furrow is just outside the knobs which are formed where the ribs and cartilages unite. The furrow extends further down on the left than on the right side; but it is deeper over the fifth and sixth ribs on the right than on the left side; the heart and the liver respectively supporting, to some extent, their corresponding ribs. The points of maximum recession correspond to the fifth, sixth, and seventh ribs. The chest expands again considerably a little below the level of the nipple, the chest walls being borne outwards by the liver, stomach, and spleen. At each descent of the diaphragm during the act of inspiration, the ribs recede where they are softest, at the part where ossification is deficient, and the furrows on the chest, described by Sir W. Jenner, are the consequence; and, just in proportion as the ends of the ribs are forced inwards, so is the sternum carried outwards. It is also a characteristic sign of the deformity in the rickety thorax, as Sir W. Jenner has pointed out, that the line of recession corresponds to the upper margin of the river, spleen, and stomach, these organs preventing recession during the act of inspiration. The precordial region thus also apparently bulges, and the chest-walls covering the heart do not recede so much as on the opposite side. Therefore, the left side appears much fuller than the right, and the precordial region appears abnormally full. (Jenner.)

The curvatures of the humerus, clavicles, ulna, and radius, require some notice. Although the bones of the upper extremity are said to be much less frequently curved than those of the lower, still the humerus is sometimes bent at an angle just where the deltoid is inserted; simply in consequence of the weight of the arms bending the softened bone when the limb is raised by the action of the deltoid. The curvature of the humerus is still further increased by the efforts which the child makes to support itself by the aid of its arms while it sits. It rests on the open palms of its hands, and thus throws a large share of its weight off the trunk on to the bones of the arm and the forearm. Thus also the radius and ulna come to be *twisted* by pronation of the palms as well as curved outwards.

After the bones of the lower extremities, the clavicles are the bones next frequently deformed. They are the subject of extreme angular curvature, mainly at two places. The greater curvature is always at the spot just outside the part where the *sterno-cleido-mastoideus* and the *pectoralis major* muscles are attached. The lesser curvature is about half an inch from the acromio-clavicular articulation. The first curve is forwards and somewhat upwards; the second curve is backwards. These curves are produced partly by the weight of the arms on the humeral end of the clavicle—the sternal end being supported by the muscles just mentioned, and by its ligaments. But the main agent in effecting the curves of the clavicle is the force which bears upon it when the weight of the trunk is thrown on the upper extremities, as the child sits with the palms of its hands resting on the ground; or as it crawls about on the floor. (Jenner.) Many of these deformities are thus traceable to the manner of carrying or placing the child, or to the weight of its body, acting on different parts of the softened skeleton, when the child attempts to stand, walk, or sit, or when it crawls about on its hands and knees. Some deformities, again, like those of the thorax, are mainly influenced by the acts of respiration and atmospheric pressure, counterbalanced by the pressure outwards of the intra-thoracic and abdominal solid viscera. (Jenner.)

The flat bones, like those of the head and the growth of the teeth, the scapula, and the pelvic bones, are also more or less affected, and give rise to characteristic deformity in Rickets.

The deformities of the head in Rickets are thus distinguished by Sir W. Jenner:—

"1st. By the length of time the anterior fontanelle remains open. In the healthy child, it closes completely before the expiration of the second year. In the rickety child, it is often widely open at that period.

"2d. By thickening of the bones. This is usually most perceptible just outside the sutures—the situation of the sutures being indicated by deep furrows.

"3d. By the relative length of the antero-posterior diameter of the head.

"4th. By the height, squareness, and projection of the forehead.

"The first two of these peculiarities of the rickety head are the result of the affection of the bones: the last two are due chiefly to disease of the cerebrum."

As a whole, the head of the child in Rickets is generally unusually large, the vertex flattened, and the forehead prominent, broad, and square, with considerable expansion at the centres of the parietal bones. The sutures also are sometimes expanded, or they remain open. The forehead, however, seems to project more than it really does, in consequence of the arrest of growth of the bones of the face and expansion of the frontal sinuses or of the nasal and ethmoidal cavities. The bones of the upper jaw, and the malar bones, are also arrested in their growth, while the under jaw appears elongated.

The process of dentition is invariably arrested or delayed in rickety children; and if the teeth have formed, they soon decay; or they early fall from their sockets. Sir W. Jenner has seen the incisor teeth fall from the jaws before the second molars of the first set had made their way through the gums. So important is the knowledge to be got from the progress of dentition, that Sir W. Jenner lays down the following rule of practice, namely:—

"If a child pass over the ninth month without teeth, you should carefully inquire for the cause. It may be that an acute illness has retarded dentition. It may be (but this is very rare) that there is some condition of the gum which interferes with the advance of the teeth. It may be (and this is infinitely the most common cause of late dentition) that the child is rickety; fail not then, when called to a child in whom the teeth are late in appearing, to look if it be rickety, for if you do fail to look for Rickets, you will most likely attribute to the irritation of teething symptoms which are the consequence of the rickety diathesis—the late dentition in Rickets being in itself merely a symptom of the general disorder. The rickety deformities may be very trifling, and yet the teeth considerably retarded in their development." (Medical Times and Gazette, April 7, 1860.)

The scapulæ are in a few instances so deformed as to embarrass more or less the movements of the shoulder. (Copland.)

With regard to the pelvis, its form varies in rickety children according to the direction in which its component bones are compressed by the spine and superincumbent parts on the one side, and the heads of the thigh-bones on the other. The direction of the forces which are thus constantly influencing the form of the pelvis and shape of its component bones, vary as the child is the greater part of its time lying, sitting, crawling on all-fours, walking, or shuffling along on the floor. On the other hand, the form of the pelvis varies also according to the age of the child when the compressing forces are brought to bear on the walls of the pelvis, and the consequent differences in the degree of ossification of the pelvic bones—the cartilages being less yielding than the bones. The rickety pelvis is therefore extremely variable as to shape, and is much more frequently triangular than oval. (Jenner.)

Next to the deformity of the thorax, that of the pelvis comes to be of the greatest practical importance, especially in the after-life of the female, relative to the functions of generation.

The sacrum and pubis may be carried either backwards or forwards; the ilia may be directed inwards, or otherwise altered; the lower part of the sacrum may be pushed upwards; and the outlet of the pelvis may thus be variously altered in form, and diminished in its diameters.

The progress of deformity, in relation to the order in which the individual bones are affected, has been attempted to be laid down by Guérin; but when it is understood that Rickets is a constitutional disease, and therefore one affecting the whole system generally, it will readily be seen that the bones are affected as one organ, and are variously deformed according to the predominating influence of the circumstances already mentioned. No one bone, therefore, is ever affected without all of them suffering; and the deformities may manifest themselves by enlargement of the ends of the bones, or by softening of the bones, or both, in a variable degree. If a child is the subject of Rickets before it walks, the ribs, clavicles, and upper extremities become deformed; while the tibiæ escape bowing, unless the child sits so as to press upon them. (Jenner.)

In a child suffering from Rickets, the large size of the wrists is generally the first deformity which attracts attention. The costal ends of the ribs, the malleoli at the ankles, the olecranon process of the ulna, and the ends of the long bones generally, are all similarly enlarged. This enlargement of the ends of the bones, and the softening, however, do not progress in an equal degree, either condition being often out of proportion to the severity or progress of the other; and Sir W. Jenner has also observed, that it is not uncommon to see the thoracic deformity lessen at the time the legs are bending—a circumstance which he considers due to the disease having greatly diminished, and the muscular power having increased, so as to permit of the child walking before the bones of the leg are strong enough to bear the weight of the body. (Med. Times, April 7, 1860.)

The extreme deformity which results from Rickets is not only very common among the poor, but it is not so uncommon as has been supposed among the rich. (Jenner.) "All degrees of softening of the bones may be seen, from that in which the ribs only yield to extraordinary forces (as during bronchitis, and then only sufficiently to flatten the antero-lateral aspect of the chest), to that in which the ribs yield at every inspiration; all degrees of enlargement of the ends of the ribs and of the long bones—from that where one might maintain that the enlargement was only that proper to the child, to that in which the projections on the anterior wall of the thorax and the enlargement of the wrist would strike the most careless observer." (Jenner, loc. cit.) The consequences of the bone-disease are often attended with extreme distress to the child, and are thus described in the eloquent word-painting of Sir W. Jenner: "It is strange to see a little child sitting placidly on the bed, without moving for hours together,—its legs placed so as to escape pressure, its spine bowed, its head thrown backward, the chief weight of its body cast on its arms; and to know that, notwithstanding the apparent calm, the tiny thing is indeed fighting the battle of life: for it is striving with all the energy it has to keep in constant action every one of its muscles of inspiration—endeavouring so to supply the mechanical defects of its respiratory apparatus, due to the softening of the ribs. It wants no toys. It is the best of children if you only leave it alone; move it, and you inflict pain on its tender frame; show it the horse or the doll that was once its delight, and it turns away its head or stares vacantly: to notice would divert its attention too much from the performance of those respiratory movements which are essential to its existence." (Med. Times and Gazette, 1860, p. 416.)

Amongst the phenomena which are significant of Rickets, the condition of the intellect has been generally described as precocious. Both Copland and Jenner, however, agree that such is not always the case. On the contrary, the child continues dull, taciturn, or stupid, or even idiotic; and, in children who are the subjects of extreme Rickets, intellectual capacity and power are always deficient; and the mental, like the muscular power, retrogrades as the constitutional ill health of Rickets continues to progress. The error regarding precocity of intellect is variously explained away. Thus, on the one hand, it has been believed that the openness of the sutures—by allowing the circulation within the cranium, and the development of the brain, to advance unimpeded, and even at an increased rate—has permitted the faculties of the mind to expand unduly; whilst on the other hand, the closing of the sutures, and the consequent unyielding state of the cranial bones, has been thought to confine and embarrass the growth and functions of the brain, and so occasion deficiency of the intellect rather than precocity. But again, according to the experience of Dr. Copland, precocity of intellect has not always been found to exist in Rickets in connexion with openness of the sutures, nor has stupidity been concurrent with their closure. Sir W. Jenner's explanation is, perhaps, the more satisfactory. Speaking generally, he believes that the mother's opinion must be well weighed before it is received as correct. She is apt to believe her child is very clever—quite a prodigy—when it is only a few degrees removed from an idiot. "The rickety child, separated, in consequence of its physical defects, from other children, and thrown necessarily much into the society of adults, catches their tricks of expression, their phrases, and even perhaps some of their ideas; and hence is thought, by the mother especially, to have a larger intellect than other children." (Med. Times and Gazette, loc. cit.)

General Symptoms of Rickets during the progress of the Bone Deformity.—The morbid conditions which existed during the developmental stage of the disease continue throughout the period when the bones soften and the body becomes deformed. The abdomen continues tumid, sometimes increases in size, and is often tympanitic. Emaciation progresses; the muscles of the limbs become flabby; they lose their power; they waste and diminish in bulk and volume. This loss of power, however, is infinitely greater than can be accounted for by the mere diminution in the size of the muscles. In proof of this Sir W. Jenner instances the case of a girl, six years of age, who was brought to the Hospital for Sick Children, in whom the loss of muscular power was so extreme that she was not only unable to stand, but even unable to support herself in the least possible degree. She lay across the arms of the person who carried her like a large half-stuffed rag doll. When placed in bed, she was incapable of changing her position without assistance; nay, she could not raise her arm an inch from the bed.

Even long afterwards, when greatly improved, she could not feed herself, and had to be tied in a chair with her head placed on a pillow at its back. If her head fell forward, the nurse had to raise it; for, unaided, she could not lift her chin from her breast. This child recovered so much as to walk about without assistance; but after her return home she unfortunately fell down stairs and was killed by the fall. (Med. Times and Gazette, 1860, vol. i. p. 416.) Although such is an extreme instance of muscular debility during the acute stage of Rickets, yet it is very common, as Sir W. Jenner observes, to see children of two, three, or even four years of age, who are quite unable to support themselves in an erect position; and if a child has commenced to walk before it has become the subject of extreme Rickets, it loses the power of walking. The child continues to become more and more languid and weak. The perspirations continue free, and are readily excited to increased flow on the least attempt merely at exertion. The thirst increases; and although the appetite is often good, the bowels are deranged, irregular, and the stools are often loose, pale or white, and devoid of healthy bile. They are fœtid, and the food is often passed as it is eaten. The pulse becomes quick, small, and weak, so that slight hectic symptoms are developed, and pains are complained of in all the bones and joints. There is complete cessation of growth, particularly of the bones. Sickness and emaciation increase, and the child appears to suffer pains except when lying on the back quite still. This stage of softening of the bones and progressive deformity may last for two or three months; and under unfavourable circumstances, when the disease is neglected, it may continue for years, the deformity slowly increasing or remaining in abeyance.

The fourth class of phenomena which may be recognised in the history of cases of Rickets embraces symptoms which may be of favourable or of unfavourable import. They are phenomena which characterise a period of restoration to health, of irremediable atrophy, or of approaching dissolution.

During this period various intercurrent affections are apt to supervene when the disease does not tend towards recovery. Under such circumstances emaciation progresses, the abdomen becomes more distended and tumid, and the bowels more disordered. The softening and deformity of the bones continue to increase, and eventually some visceral affection of the thorax or of the abdomen, or of both, terminates existence. Death occurs in Rickets mainly under one or more of the following conditions:—

(1) Intensity of the general cachexia, which, however, rarely proves fatal directly, death being in general due to one or more of the following morbid states:—

(2) Catarrh, with general congestion of the lungs, general bronchitis, or effusion into the pleura. Mechanical difficulties to respiration contribute greatly to the danger of death from these affections. "The softening of the ribs renders the mechanical power by which inspiration is performed so defective, that the impediment offered to the

entrance of air by the mucus in the bronchial tubes cannot be overcome, and collapse of large portions of the lungs follows." (Jenner.)

(3) Gastro-intestinal irritation, enlargement of the mesenteric glands of the spleen and of the lymphatic glands. The enlargement of these glands is generally the result of albuminoid (amyloid?) infiltration or degeneration; and the cachexia which is most significant of these affections is characterised by extreme emaciation and pallor of the surface of the body. Anæmia is extreme. Serum is often effused into the subcutaneous areolar tissue, and the surface of the skin has that "peculiar transparent, waxy, greenish-yellow tint which is sometimes seen in the anæmia of young women." Sometimes under these circumstances there is general anasarca—the face as well as the extremities, the hands as well as the feet, being œdematous. The deformity of the bones in such cases may be either extreme or but very moderately expressed, and may, according to the experience of Sir W. Jenner, either precede or follow the infiltration of the organs. The lymphatic glands, when so affected, vary from a small size to the size of a pea, are not tender, and never inflamed. They may be felt in the groins, in the axilla, and in the neck, rolling under the finger, and free from adhesions to surrounding tissue. They are hard to the touch, and of a globate form. The spleen is usually at the same time, with the glands, the seat of albuminoid infiltration. As its enlargement, according to the experience of Sir W. Jenner, is often overlooked, he has given the following directions for the examination of children in all obscure cases of this description:—"In every obscure case of early childhood the *absence* of enlargement of the spleen should be established. If we place the fingers of the right hand directly under the left twelfth rib, just outside the mass of the lumbar muscles; and the fingers of the left hand a little to the left of the middle line, in front and half-way between the umbilicus and the ensiform cartilage, and then press the parts forward with the right hand, and backwards and to the left with the left hand—the enlarged spleen may always be readily felt in the left hypochondriac region. We know the hard mass we feel to be the spleen, by the sharpness of its anterior margin, by the anterior margin passing from under the cartilage of the eighth, ninth, or tenth ribs obliquely downwards and inwards towards the median line. The obliquity of this line is such that usually, if continued downwards, it would cross the median line about half-way between the umbilicus and the symphysis pubis; the anterior edge is usually nearer the middle line in front in the child than it is in the adult, because in the child there is a fold of peritoneum, not usually, if at all, described in English books on anatomy, extending from the left side of the arch of the colon to the left twelfth rib, and over the anterior edge of this the spleen must pass before it can extend low enough to be detected by the hand. This fold of peritoneum causes the enlarged spleen to lie more forward, as well as to have a more oblique position, in the child than in the adult."

The enlarged spleen is still further distinguished by its moveability:

"If the enlargement be great, and the parietes of the abdomen be thin, the notch in the anterior margin can often be felt." Sir W. Jenner says that the liver is seldom so greatly enlarged but its edge feels harder and sharper to the touch than natural. In all these respects the liver and spleen correspond in their characters to what belongs to the amyloid degeneration of these organs; and, as in that degeneration also, the number of the white corpuscles in the blood is not increased.

(4) Sir W. Jenner has specially called attention to the connexion of *laryngismus stridulus* with Rickets; and that while it is an affection so constantly referred to the irritation of teething, it ought rather to be regarded as one expression of the constitutional disease now under consideration. In every case of *laryngismus stridulus* (save two) which has come within the experience of Sir W. Jenner, the child was the subject of Rickets. Dr. Wiltshire, also, in his account of "some cerebral affections of children," says that he has had abundant evidence of Rickets in cases of *cranio-tabes;* and that in such cases tetaniform convulsions were easily propagated to the respiratory muscles, giving rise to the disorder frequently known under the names of *thymic* or *laryngeal asthma.* Dr. Wiltshire also examined the body of a child who, at the age of six months, died of general convulsions following *laryngismus stridulus. Cranio-tabes* was diagnosed, and Dr. Wiltshire was able to cut off the calvarium with a small pair of scissors as easily as he could cut cardboard. The long bones could be bent like soft wax, and the bones of the skull could be folded on themselves without breaking. From the soft and yielding state of the bones, the brain is liable to pressure and other irritation; hence—

(5) Chronic hydrocephalus may supervene and prove fatal. The effusion may be within the ventricles or between the membranes, and may, or may not, be associated with tubercles on the membranes of the brain. A form of *acute hydrocephalus* has been described as due to Rickets. (Portal and Naumann.)

(6) Convulsions (*eklampsia*) may prove fatal, and nothing be found in the viscera, or within the cranium, to account for death. Hypertrophy of the brain is not uncommon; and it is often strikingly soft and anæmic. (Meric, Wiltshire.)

(7) Persistent diarrhœa may cause death; and in such fatal cases it would be well to examine the mucous surface of the intestines as to whether or not it has undergone amyloid degeneration. When the case is prolonged, and becomes chronic, none of these intercurrent affections proving fatal, the deformity continues with no abatement in the general symptoms; but oftentimes the softness of the bones diminishes, they lose their flexibility, become atrophied, and are readily broken. Deformity still progresses, recovery rarely takes place, the morbid state of the blood and the general cachexia increase, and death usually follows from some structural changes in the internal organs.

Such are the phenomena of unfavourable import, and which characterise the progressive stage of Rickets, terminating in an irremediable atrophy, or in death; but there is another set of phenomena which characterise a period of restoration, and which are of favourable import.

Such favourable changes are mainly indicated by the condition of the excreta. "The urine assumes a more natural appearance and composition; the stools are more healthy, and coloured more deeply by bile; the abdomen appears less tumid and less tympanitic; the pulse is less frequent, and pains in the limbs are not so much complained of. The countenance presents more animation, and the hectic or remittent febrile symptoms, and thirst, subside gradually. The appetite is less capricious, and more natural; and with the continuance of these changes, the flesh becomes firmer, and voluntary motion is made with greater activity. The growth of the limbs, which had been suspended till now, proceeds with remarkable vigour; the bones are gradually restored; and, if the deformity is not very great, it disappears by degrees; the curvatures are either diminished or altogether removed; the swellings of the epiphyses of the bones subside, and ossification proceeds with great rapidity—the affected bones acquiring greater density and strength than usual. The muscles also acquire a more powerful development, so that persons who have been rickety in childhood have afterwards become remarkable for strength." (Copland's Medical Dictionary, vol. iii. p. 645.)

The urine in Rickets:—The earthy phosphates (constituents of bone) have been found in greatly increased amount, both by Lehmann and Beneke, in cases of Rickets—a phenomenon no doubt connected with the increased metamorphosis of bone. "It has not yet been shown, however," writes Dr. Parkes, "that this is universal, and Rickets may be connected sometimes with simply deficient ingress of the earthy salts, without altered egress." The urine is commonly pale; and, according to Dr. Copland, the urea and uric acid are diminished, while the salts are increased. A free acid has been sometimes observed—said to be phosphoric—and a considerable sediment of oxalate of lime is not uncommon, while urinary calculi are frequent in rickety children. The increase in the fixed salts is most considerable during the advance in the first stage, and when the deformity begins to appear in the bones. It is less remarkable when the disease is far advanced, and when the softenings and flexures are the greatest. The phosphate of soda and the earthly phosphates are then most abundant. (Copland.) Lactic acid has also been found by Marchand, and by Gorup-Besanez, associated with lactates and a great excess of the earthy phosphates. An hypothesis has been raised on these observations, namely, that "an undue amount of lactic acid in the body dissolves and carries out the earthy salts." In a case recorded by Mr. Solly (Medico-Chirurg. Trans. vol. xx. p. 448) three or four times the usual amount of phosphate of lime existed in the urine. (Parkes, Copland.)

These phenomena have been described with reference to the extreme manifestation of the disease, but far less intense forms of the constitutional affection and disease of the bones are frequently seen. Enlargement of the carpo-radial epiphyses and curvature of the tibia are sometimes accompanied with so plump and fresh a condition of the child, that it is with great difficulty the parents can be induced to think there is anything amiss with it. (Wiltshire.)

DIAGNOSIS.—The constitutional manifestations of Rickets are to be recognised as distinct from those of scrofulosis, tuberculosis, or syphilis; although they have by some been all regarded as mere modifications of one and the same disease. To determine whether similar maladies are identical, four things are essential to be known:—(1) a knowledge of causes; (2) a knowledge of symptoms; (3) a knowledge of the effects of remedies; (4) a knowledge of the morbid appearance of the diseases in question. On comparing, therefore, the phenomena of Rickets in these respects with the corresponding phenomena in each of the diseases mentioned, it will at once be seen how different Rickets is from either of those affections. The general phenomena of each disease respectively are perfectly distinct from the phenomena of the others; and the pathological tendencies of each are dissimilar. As shown also by the investigations of Sir W. Jenner, the pathological tendencies of any one of these affections are rarely manifested by those who are the subject of either of the others; and although Rickets does not by any means exclude tubercle, yet it is absolutely unfavourable to tuberculization. Nevertheless, rickety children may be tubercular, just as syphilitic children may be so. Tuberculosis, also, is well known to be hereditary; but from what has been already stated under the *causes* of Rickets, it does not appear that Rickets is hereditary in the sense that phthisis is hereditary. The facts collected by Dr. Edwards and Sir W. Jenner point to the conclusion that, while more than forty per cent. of tuberculous children are born of phthisical parents, about nine per cent. only of rickety children come of phthisical parents. The converse, also, may take expression in the fact that the children of phthisical parents are not found to be especially prone to Rickets or to scrofulosis; and although several members of one family may be the subjects either of Rickets, or of tuberculosis, or of scrofulosis, it is comparatively rare for members of the same family to be the subjects of more than one of these constitutional affections. Lastly, as will appear from what has gone before, and from what is to follow, the cause, the prognosis, the pathology, the morbid anatomy, and the treatment of each of these affections are different. (Jenner, Trousseau, Merei.) Dr. Wiltshire, however, entertains a modified view, inasmuch as he regards Rickets and tuberculosis as two distinct forms, or local manifestations, of one general dyscrasia—namely, the scrofulous. (Med.-Chir. Rev., July 1856, p. 75.)

From what has been written, it may be conjectured that the diagnosis of Rickets in the precursory or incubative stage of its development will mainly lie between tuberculosis, especially of the lungs, the peritoneum, or of the brain, or cerebro-spinal membranes. A careful observation of symptoms, of records of temperature, as contrasted with those given by Dr. Sidney Ringer[1] relative to the recognition of tuberculosis, will soon show whether or not the phenomena are similar to those described under the symptoms of Rickets. When the sternal extremities of the ribs begin to swell, and become rounded in a club-shaped form; when the softened sternum begins to project more than it ought; when, with increasing pallor of the skin and increasing flabbiness of muscle, the carpal epiphyses of the radius and ulna become enlarged, and greatly so in proportion to the metacarpal epiphyses of the wrist bones, so as to give a "knotted" or "double-jointed" appearance; then the disease may be surely recognised.

Osseous tuberculosis is distinguished from osseous rachitis in the following respects:—

The bones in both diseases are loaded with morbid blood; but the blood in Rickets differs from that in tuberculosis; it is of a brown rather than of a bright-scarlet hue, and resembles chocolate rather than blood.

The modifications in the form and direction of the bones are dependent upon the weight and pressure they have to sustain.

The state of the bone is in a great measure dependent upon a deficiency of earthy material, so that the bones are uniformly soft.

The bones tend to produce regular curves, as in the spine and thigh-bones; and the pelvis becomes peculiarly distorted. The heads and necks of the thigh-bones become depressed. The joints become distorted from a yielding of their ligaments. These deformities of the bones and joints ascend progressively from the lower to the higher parts of the skeleton—the lower limbs first yielding to the superincumbent weight. (Stanley.)

There are peculiarities also in the individual bones. They tend to form exostoses. The development and nutrition seem to be vitiated from a peculiar cause in rachitis, different from that in tuberculosis.

Rachitis and tuberculosis are rarely associated; and tubercle is less frequently found in children who have died from Rickets, than in those who have died from other diseases. (Ancel on Tuberculosis, pp. 31, 32.)

A diagnosis is also to be established between the true Rickets and that softening of the bones in adults to which the name of *osteomalachia*, or *mollities ossium*, has been given. In true *Rickets*, if the patient survives, re-ossification or hardening of the bones is certain to take place; but *osteomalachia* is never followed by re-ossification. It rather induces fatty degeneration of the bones (never seen in Rickets), and irresistibly progresses to a fatal termination. (Vogel.)

PATHOLOGY. — The history of the development of Rickets is the history of the development of a constitutional disease. Such a disease becomes developed under the influence of agencies *within* the body itself, and acting through the continuous exercise of its functions. The disease is thus *inbred;* the constitution or the original organization of the child being of such a kind that the continued exercise of its functions, in place of preserving the growth and development of the body in a healthy state, becomes associated, in the first instance, with slight deviations from the standard of health, and ultimately leads to well-marked diseases, often of so fixed a character and so strongly expressed, that the local lesions have sometimes been looked upon as the real disease. Hence Rickets has so frequently been classed among diseases of the bones, and described with reference to softening of the bones only and alone, which is a mere expression (and only one expression) of a general morbid state which implicates the whole system and constitution of the body. To classify Rickets with disease of bones is therefore a mistake; for, as Sir W. Jenner has well observed, "Rickets is no more a disease of the bones, than is typhoid fever a disease of the intestines. Rickets leads to disease of the bones in the same way that typhoid fever leads to disease of Peyer's patches. The phenomena therefore which characterise the condition of Rickets, are of such a kind that they are an expression of the unhealthy state of the system, which pre-exists the development of the local lesions; and a cachectic state, a "bad habit of body," is invariably associated with the development of the disease. The change in the bones is a mere expression, and only one of many anatomical signs or characters of Rickets; just as the changes in the joints or white tissues are mere expressions of rheumatism, each of which may be regarded as so many anatomical signs or lesions developed under the influence of the constitutional disease.

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[1] See "On the Temperature of the Body as a Means of Diagnosis in Phthisis and Tuberculosis," by Sidney Ringer, M.D. 1865.

The lesions characteristic of Rickets are rarely limited to one part or system; and before death ensues, or even before the constitutional disease abates, several organs, systems, or tissues become diseased or degenerated.

In all the constitutional diseases of children (as well as of adults), the seats of such lesions or structural change are characteristic of the particular constitutional affection; while, for a long time before local lesions of structure are expressed, various forms of ill-health denote the constitutional affection.

There are several well-marked diseases of childhood which are thus constitutionally developed; namely, *rickets, tuberculosis, scrofulosis,* and *inherited* or *congenital syphilis.*

Each of these constitutional diseases is characterised by certain premonitory phenomena which taken singly, may not justify suspicion, but which, when considered collectively, yield presumptive or circumstantial evidence that certain constitutional phenomena will terminate in the characteristic lesions of structure in systems or organs of the body, which are peculiar to each of these diseases.

The deviations from the standard of health in each case may be so slight, that it may not seem to merit the name of a disease; but when these deviations are invariably succeeded by the expression of certain well-marked pathological tendencies, the relation of such slight deviations from health to such local manifestations of disease can no longer be mistaken, ignored, or overlooked.

For example, when the condition of the skin is such that it can merely be regarded as delicate, no definite diagnosis can be come to; but when we find it so often associated with the growth of tubercle in certain parts, we cannot ignore the pathological connexion between the slight deviation from the standard of health experienced by the delicate skin, and the morbid tendency of the constitution expressed by the development of tubercle in certain organs. Again, when the complexion merely excites attention by the thickness and coarseness of the features, no definite diagnosis may be pronounced; but when we know that such complexions are often associated with a peculiar form of ophthalmia, we cannot overlook the pathological connexion between the slight deviation from health expressed in the complexion, and the morbid tendency of the constitution expressed by the ophthalmia of scrofulosis.

Again, when the skin attracts attention from the mere muddiness of its tint, no definite diagnosis may be arrived at; but when it is seen that such muddy skins are frequently associated with characteristic lesions in the skin and mucous membranes, we cannot fail to connect in pathological relationship the deviation from the standard of health expressed in the tint of the skin, and the morbid tendency of the constitution expressed by the lesions of the dermis and mucous membrane, peculiar to syphilis.

So also, when the muscular power of a child attracts attention from its feebleness merely, no definite diagnosis may be warrantable; but when such lowness or feebleness of the muscular power is followed by softness of the bones, the pathological relationship cannot be overlooked, which obtains between the slight deviation from health expressed by the feebleness of the muscular power, and the morbid tendency of the constitution expressed by the softened bones.

The leading features of such typical pathological relationships as subsist between slight deviations from the standard of health, and peculiar deviations from the healthy structure of particular organs, have been thus concisely expressed by Sir W. Jenner, in the following tables:—

LEADING FEATURES IN TYPICAL CASES OF

TUBERCULOSIS.	SCROFULOSIS.	RICKETS.	SYPHILIS.
Nervous system highly developed; mind and body active; figure slim; adipose tissue small in quantity; organization generally delicate; skin thin; complexion clear; superficial veins distinct; blush ready; eyes bright; pupils large; eyelashes long; hair silken; face oval and good-looking; ends of long bones small; shafts thin and rigid; limbs straight. Children, the subject of tuberculosis, usually cut their teeth, run alone, and talk, early.	Temperament phlegmatic; mind and body lethargic; figure heavy; skin thick and opaque; complexion dull, pasty-looking; upper lips and alæ of nose thick; nostrils expanded; face plain; lymphatic glands perceptible to touch; abdomen full; ends of the long bones rather large; shafts thick.	Mental capacity and power small; muscular force deficient; mind and body inactive; figure short; closure of the fontanelles retarded; face small, but broad; skin opaque, often set with downy hairs. Children, the subject of Rickets, are late in cutting their teeth, in running alone, and in talking; and their teeth drop early from their sockets.	Adipose tissue small in quantity; muscles flabby; cutis rough, deficient in contractility; complexion, muddy.
Leading pathological tendencies.—Fatty degeneration of liver and kidneys; growth of tubercle, and consequences thereof; inflammation of serous membranes.	*Leading pathological tendencies.*—Inflammation of the mucous membranes of a peculiar kind; so-called strumous ophthalmia; inflammation of the tarsi; catarrhal inflammation of the mucous membrane of the nose, pharynx, bronchi, stomach, and intestines; inflammation and suppuration of the lymphatic glands on trifling irritation; obstinate diseases of the skin; caries of the bones.	*Leading pathological tendencies.*—Softening of the bones; enlargement of the ends of the long bones; thickening of the flat bones, and deformities consequent on these conditions of the bones; so-called hypertrophy of the white matter of the brain; chronic hydrocephalus; pulmonary collapse; laryngismus stridulus; convulsions; albuminoid infiltration of the liver, spleen, and lymphatic glands.	*Leading pathological tendencies.*—Suppurative inflammation of the mucous membrane of the nose; ulceration of the mucous membrane of the nose and of the lips, mouth, throat, and anus; falling of the hair; eruptions on the skin of peculiar character; induration of the liver; suppuration of the thymus, lungs, &c.

Rickets, therefore, can no longer be regarded merely as a local disease, characterised by a mere chemical abnormity of the bones—a mere deficiency of their earthy salts. Not only is there an insufficient deposition of the lime-salts in the growing extremities of the long bones, but the amount which is deposited there is abnormally placed as regards the anatomical constitution of bone. It is found in the cartilage cells, instead of in the matrix. And not only is there a deficiency of the lime salts, and an abnormal position of those present; but if the bones were hard before the disease began, they begin to soften, in consequence of the absorption of the lime from the shafts of the long bones, and from the substance of the flat bones. The earthy matter in a soluble form enters the blood, and is excreted by the urine. Moreover, in Rickets the growth of bone is abnormal, irrespective of the absence of lime; and the agents concerned in the nutrition of the bones, remove the lime from them, in place of taking it to them from the blood. (Jenner.)

The excessive formation of acids in the stomach of the child, has been assigned as the mode in which the phenomena of Rickets are brought about. It has been supposed that a superabundance of acid thus finding its way into the blood, facilitates the removal of the earthy salts from the bones; and it is fully proven that in Rickets, as in most other constitutional diseases, a superabundance of free acid is constantly generated in the *primæ viæ.* The particular acid is the subject of various statements. *Lactic acid* (Marchand); *oxalic acid* (Beneke, Ure, Schmidt); *phosphoric acid* (Weatherhead); and *hydrochloric acid,* have each in their turn been deemed the peccant agent in bringing about the characteristic lesion in Rickets. But all children who suffer from the generation of free acid, are by no means sufferers from Rickets. None of the acids have been demonstrated in the blood: and altogether, there are "no facts which remove the theory from the category of pure hypothesis." The theory is also in opposition to the fact that the lime is deposited in abnormal situations.

Morbid Anatomy.—A peculiar morbid condition of the bones is the anatomical sign of Rickets, just as the growth of tubercle is of tuberculosis, or as lesions of the kidney with anasarca are of Bright's disease, or as peculiar affections of the joints are of gout and of rheumatism. The most constant and striking anatomical lesions in Rickets are thus enumerated by Sir W. Jenner:—

"(1) Enlargement of the ends of the long bones,—of the parts where the bone and cartilage are in contact, *i.e.* where the cartilage is preparing for ossification, and where ossification is advancing in the cartilage.

"(2) Softening of all the bones.

"(3) Thickening of the flat bones, *e.g.* the bones of the skull, the scapulæ" [and the pelvic bones].

"(4) Deformities which follow as mechanical causes acting on the softened bones, *e.g.* the deformities of the thorax, pelvis, spine, and long bones.

"(5) Arrest of growth, not only of the bones, but of all the parts directly related anatomically and physiologically to the bones, *i.e.* of the muscles, vessels, nerves, and teeth.

"(6) Certain lesions of the pericardium, lungs, and capsule of the spleen—the direct consequences of the thoracic deformity.

"(7) Less constant but highly important changes, most commonly affecting the nutrition of the brain, spleen, liver, lymphatic glands, and muscles, and now and then of every organ."

The enlargement of the growing ends of the long bones is an actual hypertrophy. Sir W. Jenner has measured the circumference of several wrists, in rickety and non-rickety children, and he has found that, whether reference be made to the age, or to the height of the children, or to the length of the forearm, the circumference of the wrist was always greater in the rickety than in the non-rickety. The measurements of three children gave the following results:—

Disease.	Age.	Height.	Length of forearm.	Circumference of wrists.
Rickets . . .	4 years	30 inches	4½ inches	4⅜ inches
Rickets . . .	3 years 2 months	30 ,,	4½ ,,	4½ ,,
Tuberculosis .	3 years	35 ,,	5 ,,	3⅜ ,,

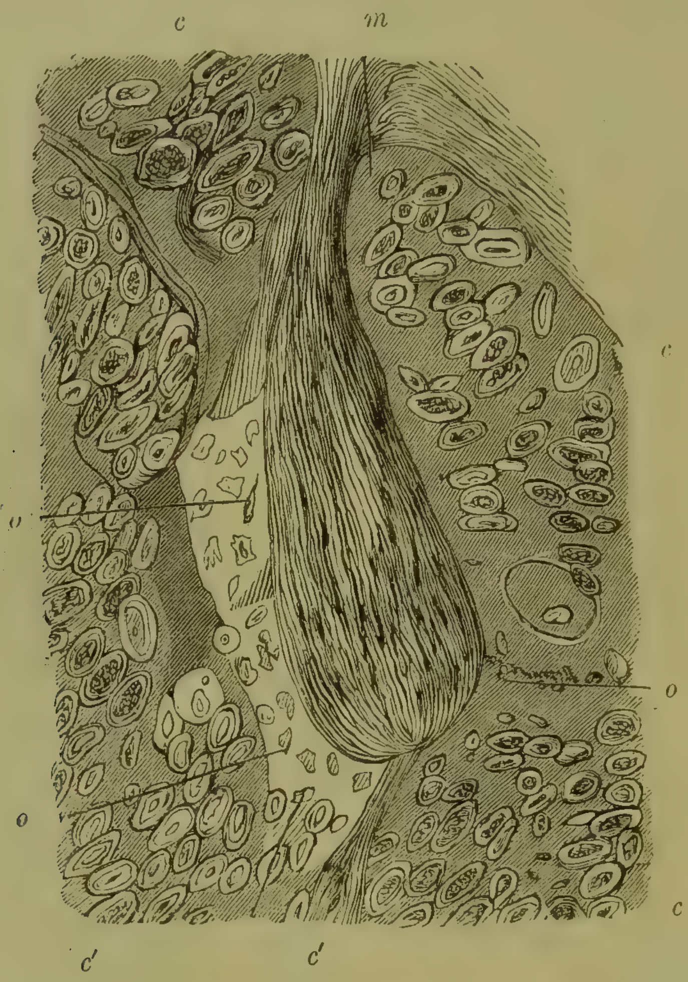
Similar enlargements are found to exist at the costal ends of the ribs, of the ankles, of the olecranon process of the ulna; and, generally, over the growing ends of all the long bones, and the bones that are the most covered by soft parts suffer equally with those which are less covered, and therefore more exposed, although the late Mr. Stanley taught that actual expansion occurs only in those joints which are superficial. In the healthy child, the ends of the long bones always measure more in circumference than the shafts, for as the processes of ossification are completed, the bone diminishes in girth.

The microscopical characteristics of the osseous substance in the bones of rickety children, have been investigated carefully by Kölliker, H. Meyer, Rokitansky, Virchow, and Jenner.

With regard to ossification in Rickets, it has been found that the anatomical characters are peculiar in the following respects: (1) in the disproportionately large epiphysal cartilages, the larger of the ossifying cartilage cells (those disposed in rows) measuring, instead of half a line, as much as from two to five lines; (2) in the border of ossification being dentated, the cartilage and the bone severally interlocking and interlacing with each other; (3) in well-marked rachitic bones, the deposition of calcareous granular particles at the border of ossification is wanting, and the cartilage cells or capsules are metamorphosed into bone cells almost invariably shortly before the matrix begins to ossify, and are also without any appearance of calcareous granules. (Kölliker.) There is thus excessive formation or hypertrophy of the structures which precede or form the nidus for ossification, while there is at the same time retardation or incomplete performance of that process. It is, in fact, an exaggeration of the condition usually seen in the first stages of ossification in the healthy subject—the completion of the process only is stayed. There is thus excessive development of the spongy tissue of the head of the bone, and of the epiphysis, and of the layer of cartilage in which the primary deposit of earthy matter takes place. (Jenner.) In the ossifying shaft of a rickety bone, Kölliker has observed that the bone cells are formed from the cartilage cells by the thickening of their wall, with the simultaneous formation in it of canalicular vacuities. If the rows of cartilage cells of the large hypertrophied ossifying border be traced from without to within, it will be seen that at the point where the deposition of calcareous salts commences, they exhibit, instead of a membrane indicated by a single tolerably strong line, a much thicker coat, which on the inner side presents delicate indentations. Kölliker believes that these cartilage cells are about to be transformed into bone cells or lacunæ. This becomes all the more evident when, further on in the growth of the bone, the thickness of the membrane, together with the simultaneous diminution of the cavity of the cell, is seen to be constantly increasing; the indentations of the interior contour line become more and more marked; and, accompanying the progress of these changes, the walls become more and more dark from the addition of calcareous matter. The very slow ossification of the matrix between the cells, in Rickets, is favourable to the observation of these changes, and permits of the alterations in the cartilage cells, and their formation into bone-cells or lacunæ, being seen, and traced step by step. Cartilage cells, enclosing secondary cells within them, are connected as a whole into a single compound bone-cell; and such cells are frequently met with having two cavities, which cells, according to their degree of development, are sometimes wide, and furnished with strong prolongations, and sometimes, from their contracted cavity and long canaliculi, resemble in all respects perfect bone lacunæ. The cartilage cells lying free, and in close apposition, though in a now ossified matrix, thus become transformed into bone-cells, with nuclei and other contents; and so an ultimate change takes place by which the bone substance in Rickets acquires pretty nearly the nature of sound tissue. These ultimate changes, so far as they affect the bone-cells, depend, in the first place, upon the commencement of ossification in the matrix, but without any primary formation of calcareous granules; secondly, upon the continuous and increasing deposition of earthy matter in it, and in the thickened cell-walls, owing to which the new bone substance becomes more and more white to the naked eye.

Kölliker's account of the lacunæ formation (of which he has given drawings) is supported by Rokitansky and Virchow. Virchow shows that the abnormity of the process of ossification in Rickets, consists "not in a process of softening of the old bone, but in the non-solidification of the fresh layers of bone as they form; and that we see the process of growth better in rickety than in normal bones, because in the rickety bones the view is not obscured by the deposit of calcareous granules. The old layers of bone are consumed by the normally progressive formation of medullary cavities; and the new layers remaining soft, the bone becomes brittle. There is also a certain irregularity in the growth of the bone, so that stages in the development of bone which, when the formation is normal, ought to set in late, set in at a very early period in Rickets. In normal growth, for example, the pointed processes (in which shape the calcareous salts shoot up into the cartilage) form, along the margin of calcification, such a completely straight line, that it should almost be described as mathematically regular." This condition ceases to obtain in Rickets, and the more so the greater the severity of the case. Interruptions occur in such a way that in some places the cartilage still reaches a long way down, whilst in others the calcification has mounted up to a considerable height. These uncalcified parts sometimes become so completely separated from one another, that they remain forming specs of cartilage in the midst of the bone, and surrounded on all sides by it; and that cartilage is still found at points where the bone ought long since to have become transformed into medullary tissue.

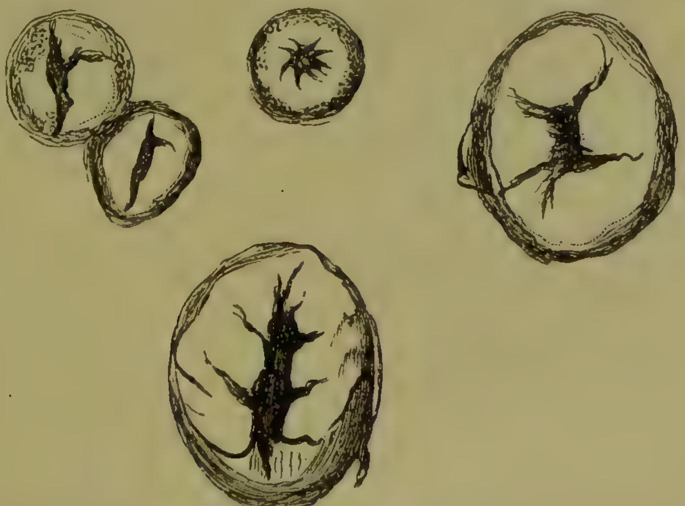
The farther the process advances, the more do we meet with isolated masses of lime scattered in the cartilage; in many instances to such a degree, that the whole of the cartilage on section appears dotted with white points. The irregularity of the process is further shown in this, that whilst in the normal course of things the medullary spaces should begin to form only at a short distance behind the margin of calcification, they exceed these limits in Rickets, and in many cases a series of connected cavities extends far beyond the border of calcification, which (cavities) are filled with a soft, slightly fibrous tissue, with vessels running up into them. Medullary spaces and vessels are therefore met with where normally and properly not a single medullary cell, and scarcely a single vessel, ought to have been found. Different histological conditions are thus found side by side, crowded into a small space: at one point cartilage, at another calcification, at a third bone or medullary tissue, and everything lying in the greatest confusion; in one place medullary tissue, above it osteoid tissue or bone, by its side calcified cartilage, and below it, perhaps, cartilage still retaining its original condition. The whole of the rachitic portion of the diaphysal cartilage acquires no real firmness, and this is one of the chief causes of the liability to distortion which rickety bones exhibit, not in the continuity of the diaphyses, but at the articular ends. This is in many cases considerable, and is the sole cause of many a deformity, as, for example, in the thorax. The curvatures in the continuity of the bones are also incomplete fractures—solutions of continuity—while those of the epiphyses are due to the proliferation of the cartilage, and constitute simple inflexions. Thus, it is easy to conceive how parts which are so entirely deprived of their regular development as in Rickets, must retain great mobility. The individual parts in the cartilage that ought at a later period to have become bone do not calcify, and the junction of medullary spaces often takes place a long way up above the border of calcification. Large and often very vascular conical processes of fibrous medullary tissue are seen, extending upwards from the bone into the cartilage. They do not force their way into the cartilage from without, but seem to owe their origin to a fibrillation of the intercellular substance of the cartilage itself. It is around them chiefly that the osteoid transformation of the cartilage can best be seen, and particularly that gradual conversion of a cartilage corpuscle into a bone-corpuscle. Out of the cartilage corpuscle, which has a moderately thick capsular membrane, arises a structure, provided with a capsule continually increasing in thickness, within which the space for the cell constantly grows smaller, and which, when it has attained a certain degree of thickness, acquires indentations on its inner wall, like the so-called dotted



Vertical section of cartilage from the diaphysis of a rickety growing tibia from a child two years old. A large conical process of medullary tissue sending out a lateral band on the left side, extends from *m* up into the cartilage; it consists of fibrous basis with spindle-shaped cells, at the circumference: at *c c c* the cartilage is in a state of proliferation, with large cells and groups of cells; at *c′ c′* commencing thickening and internal indentation of the cartilage capsules, which at *o o* coalesce and form osteoid tissue. 300 diameters. (After Virchow, p. 634.)

canals of vegetable cells. After this, a fusion of the capsule with the basis substance very generally ensues, and, with the production of anastomosing processes from the cells, the formation of bone-capsules is completed. At times, isolated osteoid cartilage corpuscles calcify alone, without the occurrence of any fusion; and whilst between them lies the ordinary intercellular substance of cartilage, the capsules of the osteoid corpuscles fill themselves completely with calcareous salts. In other places, on the contrary, the fusion of the capsules with the intercellular substance takes place very rapidly; the new intercellular substance formed by this fusion assumes a coarsely fibrous appearance, and in the place of several groups of cartilage cells, a fibrous mass is seen, containing jagged osseous (bone) or osteoid corpuscles. There is therefore no sharply-defined boundary in the tissue, but the condensed or fibrous substance which surrounds the jagged bodies is directly continuous with the translucent substance which holds the cartilage together. Essentially it is the same structure. At the point of transition at the zone where the conversion of cartilage corpuscles into a perfect osseous substance is taking place, numbers of corpuscles may be seen lying close to one another like hazel-nuts—distinguished from ordinary cartilage corpuscles by their dark contours, hard appearance, and unusually great brilliancy, and enclosing in a small indented cavity a little cell. These little cells are the bone-corpuscles—isolated because their capsules have not yet become fused with the basis substance—with calcified capsules which they have retained from that earlier period in their existence when they were cartilage cells. (Virchow's Cellular Pathology, pp. 432–436; translation by Dr. Chance.)

Sir W. Jenner does not, however, agree with Kölliker and Virchow, that in Rickets the normal process by which the lacunæ are formed is visible. His observations have led him to quite another conclusion; the calcification of the cartilage cells in the growing cartilage in Rickets, seems to Sir W. Jenner identical with the calcification of the same parts occasionally seen in *enchondromata*.



Calcification in the cells of an enchondromatous tumour similar to calcification in Rickets. (Dr. Aitken.)

Sir W. Jenner regards it as entirely a pathological process—a petrifaction. "The spongy tissue is much more spongy in appearance than natural, and from the interstices of its meshes a deep red pulp is expressible, ... composed of colourless nucleated cells, usually containing only one nucleus, now and then two, and occasionally several blood-globules, and in some cases a very large quantity of free fluid fat—evidence of excessive preparation for the process of ossification, and arrest of the completion of the process. The periosteum is thickened over the head of the bone, as it is over the bone generally, and attains its maximum degree of thickening just at the point of junction of the bone with the cartilage, and it is more vascular over the whole bone, as well as thicker. A crimson pulp fills the canal of the bone, and all the interstices of the tissue of the long bones." (Jenner.)

On reviewing those descriptions, it will be seen that the differences in appearance and in the interpretation thereof may be merely due to the various observers having examined the bones at different stages of the disease, and perhaps also in children of very different ages. This account of the morbid histology of the rickety bone also accounts for the great softness of its texture, which is sometimes so great that the bones may be bent by the least possible force, and the thick bones may be cut with a knife or a pair of scissors. Of course the softening is obviously due to the absence of those elements which render bone hard —the calcareous or earthy salts. The animal matter, also, of rickety bones does not seem to be normal; for in the experiments of Lehmann and Marchand, rickety bones did not yield gelatine on boiling. At an advanced period of the disease, Simon also affirms that "the animal matter is so changed that its extract yields on boiling neither chondrin nor gelatine." On the other hand, perfect gluten has been obtained from rachitic cranial and thigh bones, by Schlonberger and Friedleben. They did not find that the organic basis of rickety bone had undergone a change in chemical reaction, but the unossified cartilage contained much more water. Putting together the results of the analyses of several observers, Sir W. Jenner finds that the bones of healthy children yield about 37 parts of organic and 63 of inorganic matters; whereas those of rickety children yield about 79 parts of organic to 21 parts of inorganic matters.[1] Anatomical and physiological inquiries thus clearly establish the fact that a continuous osteogenetic process is going on up to a certain point only, during the acute stage of Rickets. Layer upon layer, as in health, of new matter is deposited, the deeper seated of which layers are constantly disappearing through absorption. These layers are soft and puffy; and so long as the cachexia of Rickets persists, they never become hard.

The thickening of the flat bones is similarly due to the hypertrophy of their growing portions with increased vascularity of the periosteum. There is a great abundance of nucleated cells, which, with blood, form the pulp, and occupy all the meshes and interstices of the bones;

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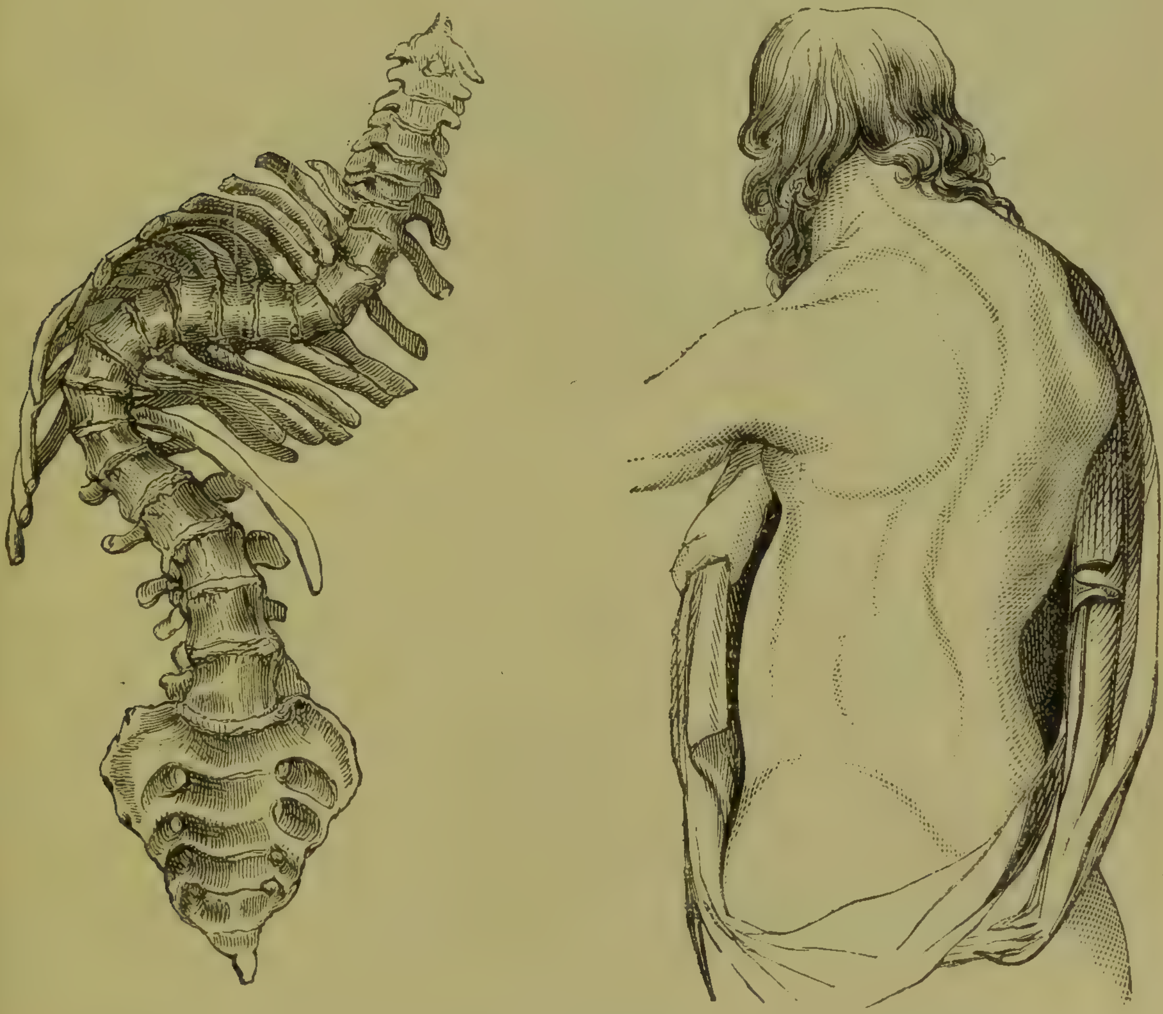
[1] See details of several analyses in Med.-Chir. Rev., July 1856, pp. 70 and 71.

so that the substance of the bone seems mainly to consist of diplöe. Next within the growing margin of the bones, and close to the sutures, the thickening of the cranial bones is usually the greatest, consequently the situations of the sutures are usually indicated by deep furrows on the scalp. So also round the posterior margin of the scapula, and at its acromial, coracoid, and articulating processes, the scapulæ are thicker than at their centres.

Hypertrophies and softenings of the bones do not always proceed in an equal degree—the softening being very often out of proportion to the enlargement, while the enlargement is sometimes out of proportion to the softening. In the rickety child the ribs are often softer than their cartilages; in the healthy and still more so in the tubercular child, the cartilages are softer than the ribs.



Permanent curvature of the spine, with rotation, produced by Rickets in early life. (Miller.)

The curvature of the dorsal and lumbar spine (the symptoms of the deformities due to which have been already described) is mainly the consequence of muscular weakness and softening of the bodies of the vertebræ. The direction of the curvature is mainly determined by the weight of the head, and is generally an exaggeration of the curvature always existing when a child of three or four months old sits unsupported on the nurse's arm. Simple lateral curvatures are thus less common than antero-posterior, with more or less rotation of the bodies of the vertebræ. These become squeezed in at the concavity of the curve on the front or side, as the direction may happen to be, while on the convexity the articular processes become commensurately both thickened and enlarged. Antero-posterior bending has occurred to such an extent as to cause doubling of the aorta, adhesion of the opposed coats at the folded part, and consequent mal-nutrition of the lower limbs. (Miller.)

When the walls of the thorax are examined from the inside of the chest after death from Rickets, the projections where the ribs join with the cartilages are much greater than on the outside; but the eleventh and twelfth ribs, which are not inflexed (as described previously), have the same enlargement in the inside as on the outside. (Jenner.) The great determining cause of thoracic deformity, according to Sir W. Jenner, is atmospheric pressure, aided by the elasticity of the lungs: and he explains as follows the mode of its operation:—"Suppose," says he, "the external thoracic parietes were made of unyielding material, then the diaphragm could descend only so far as the air could enter at the orifice of the larynx, and overcome the elasticity of the lungs. The thoracic parietes however, in their normal condition, are not absolutely unyielding, but there is a due relation between their strength, the power of the diaphragm and the rapidity of its contractions, the size of the orifice of the larynx, and the elasticity of the lungs.

"The chest-walls being healthy, and the orifice of the larynx of normal size, if the young child sobs violently—*i.e.* contracts the diaphragm with abnormal rapidity and force—the most flexible parts of the thoracic parietes will yield or fall in during inspiration.

"If the orifice of the larynx be narrowed, and if the diaphragm contract with only normal rapidity and force, there will be recession of the softer parts of the chest-walls at each inspiration. Again, if the orifice of the larynx remain normal, the diaphragm acting energetically, as in a healthy child, and the chest-walls be softened (as in Rickets), then, at each inspiration, there will be recession of the most yielding part of the thoracic walls."

These are the conditions which obtain in Rickets. The part of the rib where ossification is imperfect and incomplete, is so soft that, at each descent of the diaphragm, it recedes, and the furrows on the thorax already described at page 816, are produced. In proportion as the ends of ribs are forced inwards, the sternum is carried forwards. Sir W. Jenner rightly excludes muscular action from all direct share in the production of these deformities of the thorax or curvatures of the long bones in Rickets. He does not agree with Rokitansky that the deformity of the thorax is the consequence of any want of power of the respiratory muscles; for on dissection he finds no correspondence between the points of insertion of the muscles of inspiration attached to the outer surface of the chest-walls and the points of recession.

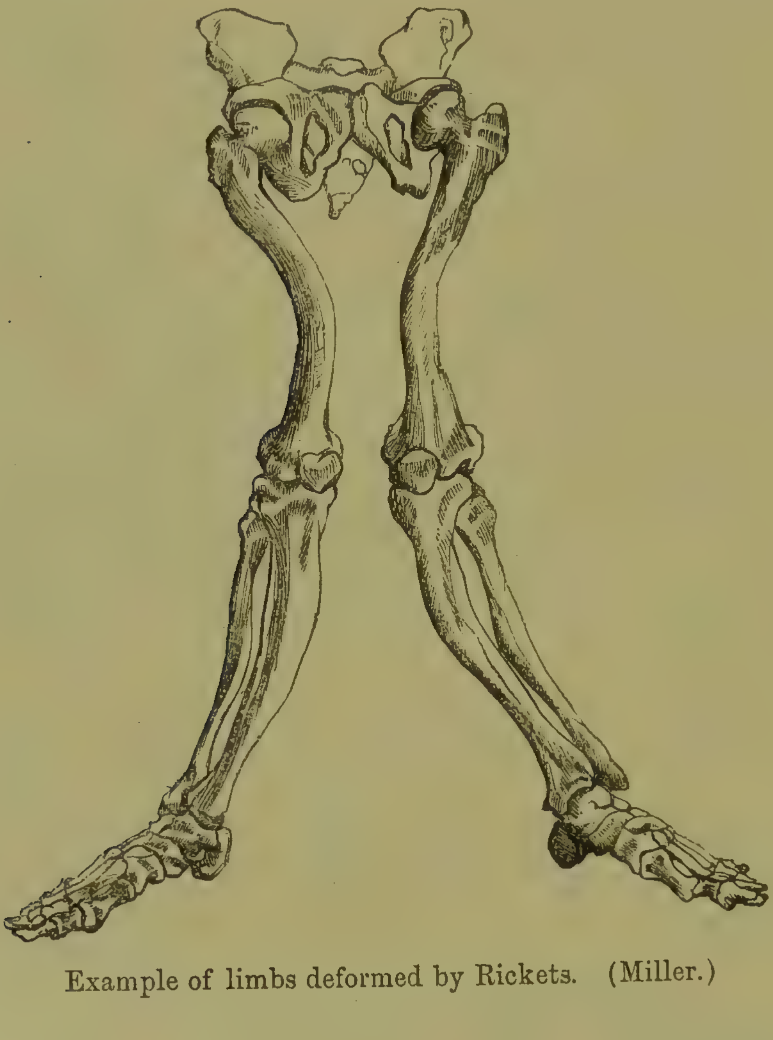
Nor does he find that the diaphragm causes circular recession by its direct contraction, as by drawing in the receding parts at each contraction; for dissections, compared with cases and models, prove that the line of recession does not correspond to the points of attachment of the diaphragm, but it corresponds to the upper margin of the liver, spleen, and stomach, and is produced (as the longitudinal thoracic furrow is) by the atmospheric pressure: the parts of the parietes below being prevented receding by these solid organs, whose influence in preventing recession of the chest-walls is illustrated by the *apparent* bulging of the precordial region in every case of well-marked rickety thorax. The chest-walls covering the heart do not recede so much as those on the opposite side; and as the left side is thus much fuller than the right, it might be supposed that there is abnormal fulness of the precordial region, which there is not.

Another point fully noticed by Sir W. Jenner, is the influence of attrition in producing "white spots" or "white patches" on the surface of the heart, in children whose chests are deformed by Rickets, and in such children the chosen seat of the "white patch" is on the *left* ventricle, a little above its apex, just at the spot which impinges against the fifth rib where it projects or knuckles inwards; for the sternum of the rickety thorax being forced forwards, the relative positions of the chest-walls and of the heart are no longer normal, and the apex of the heart strikes *outside* the nipple. "White patches," similar in nature and origin, may also be found on the spleen. They are to be distinguished from those due to embolism by the fact that they do not extend below the fibrous tunic of the organ.

In connexion with the morbid anatomy of the skeleton, the results of Mr. Shaw's observations must not be overlooked. He has shown that arrest of the growth of the bones, and of the parts in relation with them, is a very important consequence of Rickets; and the arrest of growth commences during the progress of Rickets, and persists after the general disease has ceased. Hence children are not only stunted in growth, when the subject of Rickets, but they never grow into ordinary-sized adults. Arrest of growth is most strongly marked in the legs and thighs, imparting dwarfishness to the frame, as well as distortion. While the bones of the thighs and legs are often bent in a variety of fantastic forms, they are at the same time flattened, generally so that the great diameter of the bone is antero-posterior in relation to the curve; consequently, when they ossify and harden in after-life, as the disease subsides, the limbs are not so weak as they otherwise would be. The heads and necks of the thigh-bones bend downwards, and may ultimately come to be on a lower level than the trochanters.

The articulating ligaments fail, causing deformity of the knee and ankle joints. The pelvis is small, its front wall is flattened, and forced back upon the sacrum. A characteristic hollowness is thus imparted to the loins, by the sacrum being thrust downwards; its promontory becoming usually salient, and its posterior surface forming the bottom of a hollow on the back part of the pelvis. At the same time both ilia are displaced backwards, so as to overlap the sacrum and approach each other, sometimes having scarce an inch of space between their posterior borders. (Miller.)

According to Mr. Shaw's researches, all the bones of the adult whose skeleton has suffered from Rickets are diminished in length, but the lower limbs are disproportionately diminished in size, and the face is small in proportion to the skull. This arrest of growth of the bones of the face and the sinuses causes the forehead to appear to project more than it really does. Mr. Shaw's observations were directed to the examination of the relative proportions which the different parts of the skeleton bore to each other. The relative dimensions of rickety skeletons show that they have a configuration quite different from that which belongs to the natural skeleton. (Med.-Chir. Transactions, vols. xvii. and xxvi.)



Example of limbs deformed by Rickets. (Miller.)

Such alterations in important visceral cavities, as have been described, inevitably lead to secondary lesions in the contained organs. Two lesions of the lungs are constantly present when the thorax is deformed by Rickets, namely, collapse of lung tissue and that form of emphysema which has been termed *insufflation*, due to over-distension with air of the vesicular tissue of the lung. In the lungs of rickety children its site is constant. It invariably occupies the whole length of the anterior border of both lungs, extending backwards for about three-quarters of an inch from the free margins.

The emphysematous portion is separated from the healthy portion of lung by a groove formed of the collapsed air-cells of the lung; and this groove of collapsed tissue corresponds to those projections inwards of the ribs where they unite with their cartilages.

The mechanism of the production of those lesions is thus described by Sir W. Jenner: "The softened ribs, instead of being drawn outwards at each inspiration, are forced inwards by atmospheric pressure; the consequence is, that not only are the lobules of the lung beneath not expanded, but they are compressed; and the compression of the lung, aided by its elasticity, causes the collapse."

The emphysema of the anterior border is produced thus: "The lateral diameter of the thorax is diminished at the part corresponding to the line formed by the junction of the ribs and cartilages. But at each inspiration the ribs recede; and in proportion as the ribs at this part are forced inwards, the sternum must be thrust forwards; and just as less air or no air enters into the tissue under the receding ends of the ribs, so an excess of air is drawn (as we commonly call it) into the lung tissue subjacent to the abnormally advancing sternum and cartilages of the ribs.

"The collapse is thus directly consequent on the recession of the ends of the ribs during inspiration; the emphysema is directly consequent on the thrusting forward during inspiration of the sternum."

These lesions stand in direct relation to the anatomical sign of Rickets, namely, the affection of the bones; but there are other lesions which, equally with the bone lesion, are anatomical signs of the constitutional affection, and which as such have been described by Sir W. Jenner. The emaciation of Rickets is mainly associated with the lesions about to be noticed, and which consist in a form of albuminoid infiltration of lymphatic glands, spleen, liver, brain, kidney, heart, and thymus gland. Sir W. Jenner is of opinion that these lesions are never limited to one of these organs in Rickets; but that every one of them is more or less affected, perhaps all the tissues. The cut surface of such organs is singularly pale and transparent, compact, smooth, tolerably moist, and to the unaided eye uniform in appearance. The substance is tough and the organ heavy in proportion to its size; and although Sir W. Jenner is opposed to the belief of such lesions being the same as those described by Virchow under the name of amyloid degeneration, yet with the exception of his failing to get a *blue* reaction with iodine, their characteristics appear to be similar.

PROGNOSIS.—The sooner the disease becomes established after birth the more certain is the result likely to prove fatal; but those in whom the disease appears later in life may recover before the fifth or sixth year. The general health begins gradually to improve, the tumefaction of the abdomen begins to subside, and the bones to acquire firmness. Thus becoming *set*, as it were, in their abnormal shapes, the figure always retains a certain degree of deformity. When the disease ceases, the mind, like the body, recovers, and regains all its powers. The muscles of those who were once rickety, in after-life are often marvellously powerful, their bones singularly strong, and their intellect certainly not below the average. (Jenner.) Recovery sometimes takes place after an acute febrile attack, occasionally accompanied by the appearance of a cutaneous eruption. (Cumin.)

At the approach of convalescence, an extraordinary liking for particular articles of food may be exhibited; and Dr. Cumin relates how one patient had so strong a desire for salt, that she devoured it like sweetmeats. The quantity taken was very great, and the parents believed it had proved the means of curing the child.

In severe cases, where deformity of the thorax is extreme, the great danger of death is from catarrh and bronchitis. The danger is not only in proportion to the severity of the inflammation of the air-passages, but to the degree of softening of the ribs. In estimating the danger of bronchitis in a sickly child, it is by no means sufficient to listen to the auscultatory signs, or to note the lividity of the lips, or action of the nares, or the frequency or severity of the cough, or evidence of febrile disturbance; but the child must be stripped, so as to note the degree to which the ribs are softened, how much they recede during inspiration, and to what extent they are forced outwards during expiration. (Jenner.)

If the complaint occur in children about the second year of age, or later, although it may be of considerable duration, yet amendment is generally rapid when it commences; but, even when the growth is stunted, and the deformity considerable, still the period of puberty may remarkably develop growth and diminish deformity, especially when a change of air and outdoor exercise can be enjoyed.

Generally—if the disease be not far advanced, if the child be not greatly debilitated, if the deformity have not greatly affected the spine, chest, or pelvis—a favourable result may be anticipated; but when the reverse of these conditions prevails, then complete recovery may not be expected.

THERAPEUTICS.—Looking to the nature and causes of Rickets, it is obvious that no specific remedy will cure the disease, nor will any detailed and fixed line of treatment be applicable to every case. An outline of the principles on which cases of Rickets are to be managed may be given; but the selection of particular remedies must be made for each case, according to the pathological conditions of the patient.

Improvement of the general health is the first object to be sought after. Such hygienic influences must be obtained as to secure for the patient healthy nursing, a warm and dry atmosphere, due ventilation, and pure air. The food must be selected as suitable to the respective periods of infancy or childhood, as becoming in the stages of lactation, weaning, or dentition. The diet, therefore, and the state of the digestive organs, are to be carefully seen to by the physician, If the child be under eight months old, and "brought up by hand" wholly or in part, milk diluted with about one-fourth part of lime-water, and with a teaspoonful of cream added to the half-pint of fluid, will be found the best food. It is better not to add sugar to the milk; and if it is used, it is better to be sugar of milk, and not cane-sugar. (Jenner.) Gruel, plain biscuit, aërated bread crumbs, or baked flour, may be added to the milk; and as the child gets older, beef-tea and bread, eggs and farinaceous pudding may be added to the diet. If the child be still sucking, the milk of the mother or nurse should be examined as to its quality and quantity; and, if deficient in either, the nurse ought to be exchanged for one more suitable, or the child entirely weaned, and "brought up by hand." Ass's milk has been recommended at this period; or farinaceous articles of diet should be given with mutton-tea, veal-tea, or beef-tea; or the yolk of an egg with milk may be given once or twice a day, if the child is old enough. If the child is still to be suckled by the mother or nurse, it ought to be permitted to take the breast only at stated periods; and it may have at regular intervals, in addition to the breast milk, two or three meals of milk and lime-water.

The child should be kept as much in the open air as the weather and temperature will permit. Its clothing should be warm. Its body should be washed all over, at least once in the twenty-four hours, with warm water and soap; and daily tepid or cold sponging, according to the state of the weather and condition of the child, are also beneficial. Warm salt-water bathing or sponging, or a tepid salt-water douche on the back, loins, and limbs, preceded and followed by active friction on the surface of the child, may be of service if the strength is sufficient. The child should sleep alone, and the utmost care should be taken to preserve the bed and bed-clothes clean, fresh, and perfectly dry. The sleeping room must be well ventilated; and a lamp may be burned in the chimney, to facilitate the passage of air through the room and its exit by the chimney. (Jenner, Copland.)

With regard to the administration of medicines, the physician must be guided mainly by the presence or absence of pyrexia, the state of the urine, the state of the bowels, and the progress of the cachexia generally. The irritative fever and quickness of the pulse must never induce the prescription of such lowering remedies as antimonials, nor of blood-letting. Active depletion in any form is not to be thought of; and antimony is to be held "as a poison to the subject of progressive Rickets." Mercury, unless as an aperient, in conjunction with some other drug, is equally objectionable. The fever which attends Rickets being generally characterised by asthenia, by copious or colliquative perspiration, by pale phosphatic urine, and general pallor of the skin and softness of pulse, remedies must be restorative and calmative. Alkaline remedies are considered serviceable by Dr. Copland under these circumstances, especially *carbonate of potash, liquor potassæ,* or *magnesia,* with *infusion* or *decoction of cascarilla,* or of *cinchona,* with aromatics. Lime-water charged with fixed air is useful as a drink.

If carbonates are prescribed, small doses of dilute *hydrocyanic acid,* or of the *extract* or *tincture of conium,* will be of use. If the urine be only slightly acid in reaction, or if it soon becomes alkaline, the mineral acids, especially *hydrochloric* and *nitric,* or *nitro-hydrochloric,* may be given with aromatics, or with small doses of *hyoscyamus, conium,* or of *opium.* Baths, acidulated with *hydrochloric acid,* are also worthy of use, especially if the stools are devoid of bile.

If the bowels be confined or fæcal accumulations exist, they must be relieved and evacuated by stomachic aperients, such as *compound decoction of aloes,* or equal parts of *infusion of gentian, senna,* or of *rhubarb,* with aromatics. If the stools are offensive, even if the bowels are relaxed and the stools yeasty, a teaspoonful of *castor-oil,* or of *grey powder* combined with *jalap,* may be given, and once or twice a week a small dose of *rhubarb* and *soda;* but equal parts of *rhubarb, soda,* and *calumba* powder are better than grey powder, and if continued, as they may be, with safety, the acidity of the alimentary canal will be held in check. *Prepared chalk* and *soda,* once or twice a day, may also correct superfluous acidity. When the stools are yeasty and the bowels griped, alkaline remedies should be given in lime-water and milk, with minute doses of *tincture of opium,* or of *compound camphor mixture;* and an enema containing similar alkaline ingredients may be administered. (Copland.)

When febrile disturbance has been subdued, the child must be taken as much as possible into the open air, and in the sunshine. It should as much as possible live in the open air, due regard being had to the weather, and care being taken that it be warmly clad, and not exposed to cold and damp winds. If the child has lived in town, it ought to be removed to a dry bracing sea-air, such as that of Scarborough, Lowestoft, and the east coast of England generally, in the hot months, or Brighton in the colder or foggy months. The waters of Tonbridge are well spoken of by Copland and Jenner; and although the place is inland, it has special advantages, inasmuch as while the air of the place is dry and bracing, the water from its chalybeate spa is a powerful curative agent in Rickets, on account of the iron it contains—about a quarter of a grain of the *oxide of iron* in a pint, with just sufficient *carbonic acid* to hold the iron in solution. The water of the spring has a temperature of 50° Fahr., and children will drink it readily. *Steel wine* may be added to the water, if it is desirable to give more iron. It is an extremely useful remedy, and, according to Sir W. Jenner, one of the best forms for administering iron to rickety children. A teaspoonful or two of *steel wine,* with half a grain of *quinine*, and a drop or two of *dilute sulphuric acid*, constitutes one of the best mixtures for such cases. It is especially useful when the skin is flabby, covered with perspiration, and anæmia well marked. *Tincture of perchloride of iron* in the *infusion of calumba*, or in *quassia*, or the *iodide of iron* in the *syrup of sarza*, is the best preparation of *iodine* in Rickets (Copland); and the *iodide of potassium* in a tonic decoction or infusion is sometimes also of service. A preparation new to the Pharmacopœia—the *syrup of the phosphate of iron*—possesses the general properties of ferruginous compounds, and invigorates and increases the powers of digestion. The *citrate of iron* and *ammonia* will agree with the stomach sometimes, when it will not bear more astringent preparations of iron. It is best taken during effervescence in solution of *citric acid*, flavoured with tincture of orange-peel—the salt being first dissolved in the water. (Squire.)

When the use of iron must be continued for a long time, as it is desirable it should be in cases of Rickets, *magnetic oxide of iron* is a good preparation to give twice or thrice a day. The *citrate of iron* and *quinine* is also a new and useful preparation.

The influence of *nux vomica, iron*, and *quinine* in combination ought not to be lost sight of; it is one of the most valuable of tonic remedies. The formula for its composition, as devised by the late Professor Easton of Glasgow, has been given by my friend Dr. Maclean, in his article on Dysentery; and therefore it need not be repeated here.

Any of the tonic remedies containing iron should be taken just before dinner.

Cod-liver oil is a remedy often remarkably beneficial. (Copland, Bouchut, Jenner.) It is best given immediately after meals, in orange juice or in orange wine, or as an emulsion, with milk and solution of *compound gum tragacanth.*

The condition of the intestinal discharges should be examined when cod-liver oil is given. I have often seen that much too large a dose of the oil is being given, by seeing the oil pass by the stools. If such should happen, a much less dose is sufficient; and generally, too large doses of cod-liver oil are prescribed. Superfluous acidity must be corrected by aperients and alkaline remedies, as already indicated.

The food should be carefully masticated; and if the teeth are deficient, Sir W. Jenner recommends that the food should be pounded in a mortar, and potatoes especially should be most carefully mashed. Children of twenty months or two years of age suffering from Rickets, require a small quantity of meat every day, in addition to beef-tea; and milk should form for all children the evening and morning meals. Sir W. Jenner recommends that when the stools are reported white they ought to be examined, so as to determine whether or not the whiteness is due to the quantity of undigested curd contained in them. If such should be the case, alkaline remedies are to be given, and a little lime-water added to the milk; or beef-tea may be substituted for part of the milk. The liver is not to be stimulated by purgative remedies, nor alteratives given in the belief that bile is deficient.

In the inflammatory, bronchial, and lung affections of progressive Rickets, *ammonia*, with or without *ipecacuanha* and *citrate of potash*, is the great remedy to be relied on; while for the cure of *laryngismus stridulus*, the purely tonic treatment comprehended in the combination of air, exercise by friction or otherwise, and diet, with iron and cod-liver oil, as already indicated, constitute the course of treatment to be followed up with perseverance.

Mechanical appliances are not to be recommended for straightening deformities; but Sir W. Jenner has sometimes applied splints in such a way as to project below the feet for the purpose of preventing walking.

GOUT.

By Alfred Baring Garrod, M.D. F.R.S.

Definition.—1. *Regular Gout.*—A specific form of articular inflammation invariably accompanied with uric acid in the blood, and the deposition of urate of soda in the affected tissues.

2. *Irregular Gout.*—(*a*) The same specific inflammation of non-articular tissues, or (*b*) disturbance of the functions of various organs, accompanied with the same abnormal state of the blood.

Synonyms.— Of *Regular Gout.*—Podagra (*πούς*, the foot, and *ἄγρα*, a seizure); Chiragra (*χείρ*, the hand); Gonagra (*γόνυ*, the knee): the first only of these synonyms has been much employed. Arthritis (*ἄρθρον*, a joint), a term used for general gout by the ancients, has been applied also to other joint affections by both ancient and modern writers: Goutte, *French;* Gutta, *Latin;* Gôta, *Spanish;* Gicht, *German;* terms probably derived from the idea of the dropping (Gutta, a drop) of a morbid fluid into the joints—first used in the thirteenth century by Radulphus.

Of *different forms of Irregular Gout.*—Non-articular Gout, Anomalous Gout; Podagra larvata, Goutte larvée, Goutte vague; Misplaced Gout, Retrocedent Gout.

History.—Gout was well known to Hippocrates, and his account of the disease shows that he was well acquainted with many of its salient phenomena; his remarks upon the seasons of the year at which it is most likely to occur, the subjects which it more commonly attacks, the alterations in structures it induces, the probable nature of the malady, and the difficulties experienced in effecting its cure, are well worthy of careful study.

From the time of Hippocrates almost every ancient writer on medicine has made reference to the subject of Gout. Galen speaks of the difference between the times in which he lived and those of Hippocrates, in respect to the character of the subjects afflicted with Gout. Seneca also alludes to the same topic; and accounts of the disease, more or less complete, may be found in the writings of Celsus, Aretæus, Cœlius Aurelianus, Alexander Trallianus, Aëtius, Paulus Ægineta, Demetrius Pepagomenos, and others. Nearly all these authors were humoralists, and of opinion that the disease depended upon the retention of certain matters in the blood (as bile, phlegm, &c.), caused by imperfect digestion or deficient excretion; and that these humours, or even the diseased blood itself, were thrown upon the textures of the joints, and thus gave rise to the production of inflammation, and the frequent formation of tophi, or chalk-stones.

Divisions and Classification of Gout.—It is stated in the definition that Gout may manifest itself, simply, in the form of inflammation of one or more joints, or as inflammation of some non-articular structure; or by causing an alteration in the functions of certain organs; and these latter manifestations may either be independent of, or accompany the articular affection.

When the joints are solely or principally involved, the disease may be conveniently designated regular or articular gout, which may be either acute or chronic; but when severe affections of internal organs ensue, or when inflammation of tissues, other than those pertaining to the joints, arises from the presence of Gout, these affections are known by the name of irregular or non-articular Gout. The whole phenomena of the disease can be conveniently discussed under these two heads.

Description of an attack of Acute Gout, and of the progress of the Disease.—Under this heading will be included, first, a sketch of an early and uncomplicated attack of Gout, as ordinarily met with in practice; next, an account of the progress of the disease when unchecked by hygienic or medicinal means; and, this task being accomplished, an analysis will be made of the different symptoms exhibited during the paroxysm, and of any structural alteration caused by it. In many instances the first attack of articular Gout comes on without previous warning; or, if there be premonitory symptoms, they are so slight as to pass unnoticed by the patient. This absence of warning, however, is by no means so common as is usually supposed, and I have met with several cases in which the premonitory symptoms have been very distressing; although before the seizure they were not suspected of being the precursors of any joint affection. Under ordinary circumstances, an individual retires to rest in his usual health, but early in the morning, usually from two to five, awakes with an uneasy feeling, probably confined to one of his great toes; on attempting to place his foot on the ground, he finds himself unable to support the weight of his body, or, if capable of so doing, the act is accompanied with great pain.

If the painful part, generally the ball of the toe, be examined, it is found to be swollen, red, hot, and exquisitely tender, and sometimes to such an extent that the mere weight of the bed-clothes is intolerable, and even the vibration of the room causes discomfort. The veins proceeding from the toe are turgid with blood, and the joint stiff. Although occasionally no constitutional disturbance is present, yet more frequently there is evidence of slight fever; the patient has a feeling of chilliness, followed by heat of skin and perspiration, some thirst and loss of appetite, a white tongue, and confined bowels, with great restlessness, and is unable to find an easy position. The urine is usually small in quantity, high-coloured, and deposits, on cooling, a sediment varying in colour from pale buff to brick-dust red; occasionally when febrile disturbance runs high the fur which encrusts the vessel is of intense pink colour; cramps of the legs are often present during an attack, and add much to the sufferings of the patient.

If moderate precautions are taken, and the foot kept in a horizontal position, the inflammation usually subsides in the early part of the day; but at evening an exacerbation takes place, and for the greater part of the night the patient is kept awake by the pain, which again subsides as morning advances.

After a day, or as soon as the swelling increases, considerable relief is experienced, and in a few more days the tension becomes diminished, as well as the heat and livid redness, and slight sustained pressure will then cause distinct pitting. Subsequently, as the disease disappears, desquamation of the cuticle takes place, and occasionally the skin peels off in flakes of considerable size. Not all cases, even of first attacks, assume this sthenic form; in weakly subjects, and especially in women, the fit may have an asthenic character; the pain and heat may be slight, the redness and swelling by no means well marked, yet as far as ultimate mischief is concerned, this variety is often much worse than the other.

The duration of the joint inflammation varies considerably in different cases, and is much influenced by the diet and regimen adopted, and likewise by the medicines administered. If no material change is made in the diet, and no remedies taken, the inflammatory action seldom subsides under a week or ten days, and occasionally it lasts two or three weeks; but, under more favourable circumstances, the duration of the fit is usually limited to four or five days. After the complete subsidence of the joint affection, the patient not infrequently expresses himself as feeling lighter and altogether better than before its occurrence. It will be seen that the ball of the great toe has been mentioned as the joint especially chosen as the seat of the gouty seizure, and it is a remarkable fact, that a patient may experience repeated attacks of Gout in this one joint, without either the tarso-metatarsal or the phalangeal articulations being in the slightest degree implicated.

It is not an uncommon occurrence for both great toes to be attacked, even in a first fit of Gout, sometimes simultaneously, but more frequently alternately, the inflammation rapidly subsiding in one toe, and as quickly appearing in the other. Sometimes other joints, as the ankle, are affected at the same time as the toes, and occasionally the knees, or more rarely some joints of the upper extremities.

In many instances, some two or three years elapse before the occurrence of the second attack, but in the majority of cases not more than twelve months; and then either the same joint as in the first seizure, or the corresponding joint in the other foot, is usually affected. Similar intervals elapse between the next few paroxysms, and again the same joints are implicated, or the inflammation extends along the foot, involving the articulations of the arch and the ankles.

As time goes on, the disease becomes more general, and almost every joint of the extremities suffers, those of the lower usually taking precedence of those of the upper limbs. The hips and shoulders are, perhaps, less liable to be attacked than the rest, although they do not necessarily escape. In exceptional cases, other articulations, as the spine and jaw, become the seat of gouty inflammation.

In the course of years, the intervals between the attacks diminish still more—the yearly visitations become half-yearly; afterwards the attacks recur every few months, until at length the patient can scarcely calculate upon being free, so numerous and uncertain are the visitations of his malady.

Phenomena occurring during an Acute Gouty Attack.—It will be interesting to examine a little in detail the phenomena which present themselves during an acute paroxysm of Gout, for our diagnosis must be founded in part upon the peculiarities exhibited at such a time.

1. *Febrile Disturbance.*—The febrile disturbance, indicated by heat of skin, temperature of axilla, thirst, loss of appetite, and rapid pulse, is almost invariably in close relation to the number of implicated joints, and the intensity of the inflammatory action; in other words, the fever is secondary, and dependent on the joint affection. It is important to remember this fact, because it will be found that in some other diseases—for example, in acute rheumatism a patient may exhibit all the symptoms of intense febrile excitement, at a time when the joint affection is scarcely appreciable.

2. *Local Appearances.*—The appearance of the inflamed joint is usually characteristic; there is much swelling present, and enlargement of the veins proceeding from the joint, also great tension of the skin. As the inflammation subsides, pressure produces distinct pitting, indicating the presence of œdema. After a further interval, desquamation of the skin almost invariably occurs, usually in a marked degree.

There is a point connected with gouty inflammation which is not without interest—namely, the fact, that however acute in character, it never leads to the formation of pus. An inflamed joint may be intensely red, even scarlet, the skin shining from the distension, and it may altogether exhibit the appearance of suppuration; yet all these symptoms quickly subside, and by resolution merely. When it is stated that a part affected by gouty inflammation never suppurates, it should be added, unless previously the seat of chalk-like deposits; in which case it is not uncommon to find matter formed around such concretions, this formation of pus being probably due not to gouty, but to common inflammation set up around previously existing deposits, which have by their presence acted as foreign and irritating bodies. In enfeebled conditions of the system, such an occurrence is frequently met with.

The pain which attends the joint affection must not be overlooked. It is a common opinion that gouty pain is very intense, a degree more so than that arising from other articular inflammation. Doubtless this is often the case, but it sometimes happens that an acute attack of Gout may be nearly painless, the amount of suffering depending much on the rigidity of the structure of the affected articulation, as well as the peculiarities of the patient. The first attack of gouty inflammation in a joint—for example, in the wrist—may be attended with exquisite pain, but in subsequent seizures this symptom may be comparatively slight.

Œdema of the affected part has been specially mentioned among the peculiar symptoms of gouty inflammation; and although it now and then occurs in other forms of inflammation, yet is it scarcely ever absent in Gout. It is owing to the presence of the œdema that so great tension usually accompanies the swelling, and probably the subsequent desquamation of the cuticle is also partly due to it. Not only does effusion occur in the texture of the skin, but, when a synovial membrane is inflamed, a large amount of fluid is generally poured into the joints, or, when bursæ are implicated, they become rapidly filled; this copious effusion frequently causes considerable alteration of shape in the joints.

Joints affected in Gout.—In giving a sketch of the progress of Gout, allusion has been made to the order in which different joints are affected as the attacks become multiplied, and it was then stated that the great toe is commonly selected as the first seat of the disease. The extreme frequency of the selection not only of the great toe itself, but even of a particular joint of this toe, is a fact so peculiar as to make it desirable that a few lines should be devoted to the consideration of the subject.

The joint of the great toe, so commonly the early seat of gouty inflammation, is, as before stated, the metatarso-phalangeal joint, ordinarily termed the ball of the great toe: and from a table collected by the late Sir C. Scudamore, it would seem that in 512 cases of Gout, at its first seizure, the great toe was implicated in 373 cases; and in 341 out of 512 cases, one or other, or both, great toes were affected, to the exclusion of other parts. My own experience fully confirms the general accuracy of these numbers; but it should not be forgotten that this joint occasionally escapes altogether, and cases of severe Gout, accompanied with excessive deposits, have come under my care—cases of at least twenty years' duration—in which the great toes have throughout remained free from disease. The occurrence of inflammation confined to the metatarso-phalangeal joint of the great toe always conveys suspicion of the existence of a gouty habit: but it requires caution before making a diagnosis from this symptom alone, as the joint may be for a time exclusively inflamed in other and more serious conditions of the system. It is not, however, the great toe which is always first attacked; for it often happens that an injury to the knee, caused by a fall from a horse, will induce the first development of Gout in that joint, although after a short time the great toe may be likewise affected. Even an old injury will, as it were, attract Gout to the damaged part, and cause it to linger there longer than in other localities.

It is a very common remark that Gout differs from rheumatism in implicating the smaller articulations of the body. This is doubtless true, if it has reference solely to the earlier attacks, but after a time the larger and smaller joints appear to be indiscriminately affected. It is not uncommon to hear a patient calling his disease Gout as long as it is confined to the feet, but rheumatism or rheumatic Gout when the upper extremities become attacked, although the same condition of the system which causes the one gives rise to the other also.

There are certain joints of the extremities which appear to be less liable to suffer from Gout than others. Of these the hips and shoulders are the chief: still, it must not be thought that even these joints are unassailable by the disease, for they are sometimes severely affected.

After-effects of Acute Gout.—It has been stated above that, after an attack of acute Gout, especially if an early one, the patient not unfrequently expresses himself as feeling even better than before the seizure, the affected joint recovers, to all appearance, its natural size, the tenderness entirely subsides, and its power of movement is not perceptibly interfered with. To explain this improvement is not difficult. During the occurrence of the inflammation, the blood, as we shall find, loses to a great extent, if not entirely, the morbid condition which previously existed, and hence the disappearance of the malaise; and the joint, although, as will be proved, decidedly altered by the attack, may yet be not sufficiently changed to interfere with its normal functions. This favourable termination is by no means constant, for as the disease continues to make progress, and the joints have been more frequently attacked, some little remaining stiffness is commonly experienced, due in part to mischief which is irremoveable, in part to thickening of the tissues and enlargement of the vessels, which are long in recovering their natural condition.

At times, even an early attack of Gout may lead to much mischief; when, for example, the feet are allowed to remain inflamed for any lengthened period, either from want of treatment, or from treatment injudiciously applied; considerable œdema may remain long after all pain and heat have subsided—a state often requiring special treatment for its removal. This result I have several times witnessed in patients who have allowed the disease to run its own course, and also after homœopathic treatment. Occasionally anchylosis of a joint occurs even after a few attacks; in some of these cases it is probable that disease of the joint previously existed, although not in itself sufficient to produce appreciable inconvenience; in others, active and injudicious treatment, as the application of leeches to the joint, has been the cause of the mischief.

Number of Joints affected.—In our description of an attack of acute Gout, we have taken as an illustration a case in which only the metatarso-phalangeal joint, or the ball of a great toe, has been affected.

This often happens in the first seizure, and may even occur for several years in succession; but sooner or later, if the disease continues to make progress, not only are other joints implicated, but several are affected either at the same time or in the course of the same attack; and it is not uncommon to find many joints, both large and small, of the upper and lower extremities, simultaneously in a state of acute suffering. When such is the case, as the accompanying fever is in proportion to the joint affection, the patient's malady may easily be mistaken for one of acute rheumatism; and unless the history is carefully inquired into, the diagnosis is somewhat difficult.

Premonitory Symptoms of Gout.—Some of these are referable to an altered condition of the digestive organs; a form of dyspepsia is induced, and the patient experiences flatulence, often to a very uncomfortable degree, accompanied with heartburn and acidity. Many gouty subjects, from the unusual prevalence of these symptoms, can predict the advent of an acute seizure. In some the function of the lower bowel becomes altered, and either constipation or diarrhœa ensues. The character of the alvine evacuations may also be changed.

A crampy state of the muscles is another very common forerunner of a gouty paroxysm, usually in the lower extremities, and more especially in the calves of the legs.

Palpitation of the heart is experienced by some patients on the eve of a gouty seizure, and this may or may not be accompanied with dyspeptic symptoms.

In some individuals the respiratory function is implicated under like circumstances, and a species of asthma produced. At times the urinary secretion undergoes a very visible change in character; from being copious and clear, it may become scanty and turbid. On the other hand, the urine sometimes becomes unusually abundant and limpid a few hours before the establishment of the articular inflammation.

In other persons, derangements of some portions of the nervous system are produced, the temper becomes very irritable, unusual drowsiness, or headache, grinding of the teeth during sleep, startings of limbs, and various other phenomena, may be experienced.

Lastly, it now and then happens that a feeling of unusually good health, with apparent increase of both mental and bodily power, is a prelude to a gouty attack.

All these phenomena are probably dependent on the altered state of blood which always exists previously to the development of articular Gout, and the cause of the diversity of the symptoms in different persons must be sought for rather in individual peculiarities, and the proneness of certain functions to be disturbed, than in any variation in the proximate cause of such symptoms. When any organ or function is implicated in a very marked degree, a form of irregular Gout is established, which we will afterwards describe.

Phenomena of Articular Gout when it assumes a Chronic Form.—In Gout, as in other inflammatory diseases, it is difficult to draw an accurate line between the acute and chronic stages; but it is not difficult to establish a boundary sufficiently well marked for all practical purposes. It has already been remarked, that acute attacks may come on so frequently, and apparently from such slight causes, that the patient can never calculate upon being free; but when, in addition to the frequency of the attacks, their duration is prolonged, and a notable change has taken place in the structures of the articulations, the case has assumed the characters to which the name of Chronic Gout is commonly applied.

It is a somewhat remarkable fact, that although the feet and the joints of the lower extremities are usually the seat of gouty inflammation, often for many years before the hands; still the latter are frequently seriously injured when the feet have as yet escaped appreciable damage. The explanation of this tendency of Gout to cause greater mischief in one part than another, will be attempted when the pathology of the disease is investigated.

Chalk-stones, or Tophi.—The principal changes which take place in parts affected with gouty inflammation, are due to the deposition of a peculiar chalk-like matter in the different structures; and as such deposits are not only peculiar to Gout, but when capable of being seen become a pathognomonic sign of the disease, it will be interesting to describe somewhat fully their nature and origin.

White spots often appear upon the helix of the ear, and an opportunity is occasionally afforded of observing the whole train of phenomena exhibited from the commencement to the full development of the little chalk-stone. The earliest appearance presented is that of a small vesicle under the skin of the helix, as if situated between it and the fibro-cartilage; the contents of the vesicle are at first opalescent, or milky, but afterwards become white and opaque, and acquire the consistence of cream. After some months, the vesicle assumes the appearance of a small, hard, and white bead, closely resembling a pearl, and it may remain as such for years; but occasionally the thin skin is worn off, and the bead itself becomes detached from the cartilage, leaving only a slight indication of its presence. If the vesicle is punctured in the early stage, a milky fluid exudes, which presents under the microscope the appearance of a transparent liquid, in which are floating a large number of very fine crystalline needles; if the contents are examined at a later stage, the crystals are found aggregated into small bundles; if the bead is solid, it is difficult to separate them, as they adhere strongly together, and form a closely-interlaced crystalline mass; if, instead of the little chalk-white bodies in the ear, the formation of deposits in other situations is observed, very similar phenomena are exhibited.

It will be found as we proceed that although chalk-stones, or white deposits visible upon the surface of the body, are far from being constant in cases even of long-continued Gout, yet deposition of urate of soda invariably occurs within some of the structures in every paroxysm: and thus stiffening and deformity are often induced.

When the deposition is confined to the cartilages, unless very extreme, the injury to the mobility of the joint is comparatively slight; but when the ligaments are infiltrated, they are made rigid, and the play of the parts is consequently seriously interfered with. Much distortion is caused when the bursæ become infiltrated; this infiltration, at times, takes place to an enormous extent; but it may be well to state, that the amount of secretion of urate of soda has no necessary relation to the acuteness of the inflammation.

Without the aid of drawings, it is not an easy matter to give even an idea of the extent of crippling and deformity which occurs in some subjects who have suffered from severe chronic Gout. The hands become greatly altered in appearance: sometimes, when the deposits are chiefly located in the ligaments and tendons, extreme stiffening takes place, without any important amount of bulging; many of the phalangeal joints become rigid and flexed, others equally rigid but extended beyond the straight line or curved backwards: thus the metacarpo-phalangeal of a finger may be flexed, the first phalangeal joint curved backwards, and the second phalangeal articulation sharply flexed; one or several fingers may be thus affected. At other times, not only is there anchylosis of several joints, but likewise great deposition of mattter, which causes bulging at different points, from the formation of concretions more or less hardened. In extreme cases, an appearance is presented by the hand very closely resembling a bundle of French carrots with their heads forward, the nails appearing to take the place of the stalks; sometimes the toes are affected, though usually in a less degree. The bursæ over the elbows are often distended till they attain the size of small oranges; the bursæ over the patellæ may likewise become enlarged. Deposits of different sizes are found along the shafts of bones, apparently originating in the periosteum; also on the tendinous sheaths of some of the muscles; in fact, every bursa may be affected, as likewise every tendon and membranous structure, and thus no limit can be set to the deformity which chronic Gout can produce.

When external deposits are visible in any patient, no possible doubt can exist as to the nature of the case, for, as the deposition of urate of soda in the tissues occurs only in Gout, its presence constitutes a pathognomonic sign; but, as before observed, the occurrence of visible chalk-stones is not constant, and it was asserted by Sir C. Scudamore that not one gouty case in ten exhibited them. This statement, however, is not correct, for the small concretions we have described in the ear are extremely frequent, and may constitute for many years, or even during life, the only visible deposition of urate of soda throughout the body; and as they produce no amount of inconvenience, they may altogether escape notice, unless specially sought for. Some few years since, having been able in some difficult cases to make correct diagnosis from the presence of these aural deposits, the writer was induced to investigate the matter, and, in thirty-seven cases examined within a short period, they were found to be present in sixteen cases; in seven no other concretions could be seen; in nine there existed deposits around the joints; and in one case only were chalk-stones visible elsewhere without being present in the ears likewise.

Small chalk-like deposits are found in other situations than those above mentioned; sometimes they can be felt under the skin along the tendinous aponeuroses of certain muscles, especially those of the leg and thigh, varying in size from a flattened pea to a small bean; they have been observed on the sclerotic coat, likewise on the tarsal cartilage at the angles of the eyes. It is questionable if they ever originate in a very vascular tissue such as the skin, although it may subsequently become pressed upon and involved.

It has already been stated, that the original condition of these deposits is that of a liquid, rendered more or less milky or opalescent from the presence of acicular crystals; that as the fluid part is absorbed, the consistence becomes creamy, and at last a solid concretion is produced. If the effusion has taken place in a bursa, the resulting chalk-stone is free and of uniform composition; but, if it has been infiltrated in a tissue, the structure of the part becomes mixed up with it when solidification occurs: hence the discrepancies which have arisen in different statements regarding the composition of chalk-stones.

Several analyses have been made of chalk-stones which have either been removed during life, or obtained from the body after death, and from these it will be seen that, omitting the animal matter and the soluble salts derived from the structures in which the concretions have formed, urate of soda is practically the only salt which they contain. Possibly in some instances, as in a concretion analysed by L'Heretier, the phosphate of lime found in large amounts was derived from the tissue; but it is not improbable that it may be occasionally secreted as the result of common inflammation set up by the presence of the urate of soda, which latter salt has acted as a foreign body, in the same manner as tubercular matter often becomes infiltrated with bone earth.

Gouty Abscesses.—When gouty deposits increase in size and approach the surface, the skin over them becomes gradually thinner, and often gives way; a discharge takes place, either of a white solid substance, should the concretion have been of long standing, or of liquid matter, if the deposit is more recent; but not infrequently, however, a mixture of both solid and liquid chalk occurs, and thus a gouty abscess is established. Such abscesses are usually difficult to heal, and may remain open for months, and even years; and this is especially the case when the morbid matter penetrates to any considerable depth, as, for example, in the neighbourhood of joints; but when in a bursa, as that over the olecranon process of the elbow, this difficulty is not experienced, in fact the healing takes place with as much facility as in the case of an ordinary abscess. The discharge from these abscesses may be unaccompanied with pus, and consist simply of urate of soda; but in exceptional cases, when concretions have become very solid, and the patient is reduced to a very weak state of health, inflammation and suppuration may arise, and pus mixed with white fragments is then freely discharged.

It is not uncommon to find that patients, in whom chalk-stones have been freely formed, have a great number of abscesses discharging at one time, and it is astonishing to see how little disturbance of the system is produced by them. Occasionally five or six such abscesses will be open on each hand, and nearly as many on the feet; the free outlet thus given to the matter appears, in fact, to give relief to the system.

Constitutional Symptoms in Chronic Forms of Gout.—It remains, before completing the description of chronic Gout, to speak of the symptoms which accompany the changes of structure above described. It may be here assumed (a fact which will be afterwards proved) that the blood in chronic Gout is always in an impure state, and we should expect, therefore, that symptoms indicating its irritating action upon various organs would be present. This is often the case, and chronic dyspepsia, accompanied with acidity, heartburn, flatulence, pyrosis, and pain after food, are common accompaniments; the bowels are apt to be disordered, and the function of the liver impaired, palpitation and irregular action of the heart may be present, and ocasionally symptoms referable to the nervous and muscular systems, as cramps, twitchings of limbs, nervous depression, and so forth. Although the patient is apt to be afflicted with some or even several of the above-named miseries, yet it is not always the case, for it would seem that in many such subjects the system gets gradually accustomed to the impurity of the circulating fluid, and it is only when there is an excess of such impurity that they become sensible of its presence.

The urine of patients suffering from chronic Gout, with extensive deposit of chalk-stones, is generally pale, of light weight, and often contains a little albumen; the occurrence of deposits is rare, except about the time of a paroxysm of a more acute character.

Irregular Gout.—The subject of irregular Gout is one of no small difficulty, and requires careful handling in order to avoid falling into error. Some pathologists appear to assume that all the ailments which happen to patients subject to Gout necessarily owe their origin to that diathesis, and hence the descriptions given of gouty pneumonia, gouty hepatitis, and many other inflammatory affections; but a closer investigation of several so-called gouty complications has led me to believe that they are nothing more than ordinary forms of inflammatory disease, modified, indeed, to some extent by the diathesis of the patient. A man with a gouty diathesis may be exposed to cold, and have pneumonia developed from such exposure, and yet the lung inflammation may not differ in its essential character from what occurs in a previously healthy person. Such a subject is, in fact, as liable to be attacked with inflammatory disease as any other individual; it is necessary, therefore, that good proofs should be shown that a malady is truly gouty in its nature, before we are justified in coming to the conclusion that it is so. On the other hand, it is equally important that, the possibility of a disease being dependent on a gouty state, the habit should not be overlooked; for upon a correct diagnosis of such cases success in treatment mainly depends.

As yet we have only described Gout as implicating the structure of the joints, or at least tissues similar to those of the articulations, and situated near the surface; but it must be remembered that like tissues exist in the deeper-seated parts of the body, and it is a question of interest to know if they often become affected; in the case of acute rheumatism, in which inflammation of the membranes of the heart so frequently happens, such occurrences are looked upon as ordinary phenomena of the disease.

Retrocedent Gout.—There is a popular belief that a patient when labouring under gouty inflammation of any joint, if exposed to cold, is liable to have the local malady suddenly checked, and to be attacked with some acute affection of an internal organ, as the stomach, heart, or brain; and when this takes place, the term Retrocedent Gout is applied.

Gout affecting the Nervous System.—When the brain or its membranes become implicated, the symptoms may be exhibited in the form of intense pain of the head, epilepsy, &c., or the intellect may be impaired, and delirium ensue.

Apoplexy has been said to have been caused by retrocedent Gout. If a patient is suffering from any chronic brain disease liable to induce apoplexy, it can easily be imagined that the suppression of articular Gout might cause its rapid development. Serous or congestive apoplexy may possibly result from gouty inflammation; but these cases are rare: the author has not yet witnessed one which could be fairly classed under such a head; those he has seen have always been accompanied with albuminuria.

A severe form of headache is not very uncommon in Gout; sometimes it occurs prior to the development of the joint inflammation, and then it usually vanishes at once on the occurrence of the latter, and now and then the alternation between the headache and toe affection is characteristically and unmistakeably marked. At times when a patient is suffering from Gout in some joint, a cessation of the articular pain suddenly ensues; but this, far from being the termination of the disease, may be followed by delirium, attended with more or less febrile disturbance. This condition may last for hours, days, or even weeks; it may be relieved by the reappearance of the joint disease, or it may gradually disappear without such re-development.

When these symptoms arise in the course of articular Gout, to what pathological condition must they be ascribed? It would seem probable that they may be ascribed to the sudden occurrence of gouty inflammation about the membranes of the brain, or the lining of the skull; and, although I am unaware of any trustworthy post-mortem examinations proving the existence of deposits of urate of soda in these structures, still such may arise, and the proof of their existence or non-existence is a point of much interest and importance. Deposits of this kind can readily escape notice, unless specially sought for; and even those which occur in the joints in early attacks of Gout were not observed till within the last few years.

Epilepsy is not uncommon in gouty subjects, and appears to be closely dependent on the diathesis which gives rise to the articular affection; it sometimes distinctly alternates with the joint affection; at others, the two may occur simultaneously.

Mania is frequently the result of retrocedent Gout. I have seen numerous cases: Gout often leaves a joint suddenly, from exposure to cold or an intense mental shock, and after a short time distinct mania is developed; this may last for days or weeks, but generally terminates favourably.

Spinal affections, probably from inflammation of the meninges, with startings of limbs, hyperæsthesia and other characteristic symptoms, occasionally result from the sudden suppression of articular Gout, or occur along with the joint inflammation.

Acute neuralgia of different nerves is at times closely connected with Gout. Sciatica of this character often occurs, and facial neuralgia occasionally. These affections probably depend on gouty inflammation affecting the sheath of the nerves. Forms of local paralysis have also been observed apparently due to the same cause; and cramp sometimes becomes so excessively developed, and so permanent, as to justify its being looked upon and classified as a form of irregular Gout.

Gout affecting the Digestive Organs.—When the stomach is affected by gouty metastasis, which sometimes occurs from the application of cold to the extremities, there is usually sudden intense pain and spasm in the epigastrium, oppression, and vomiting of bilious matter; at times the heart's action is involved, and a feeling of great anxiety, with palpitation, produced. Many of the cases reported as examples of retrocedent Gout will not bear a close investigation; still there is no reason to doubt the possibility of such an occurrence. True retrocedent Gout affecting the stomach is probably of an inflammatory nature, though no direct proof has been afforded in support of this statement.

Sometimes the upper extremity of the digestive tube, as the pharynx and œsophagus, becomes distinctly affected in Gout, producing difficulty of swallowing; at other times the rectum is implicated, and the patient is troubled with piles or tenesmus. Constipation is very common, but, on the other hand, diarrhœa may prevail, apparently of a conservative character, and cases now and then are seen in which articular Gout appears to be warded off by the discharge from the intestinal canal.

Gout affecting the Circulating Organs.—When articular Gout suddenly recedes and the heart becomes affected, the symptoms experienced by the patient are a sensation of extreme anxiety, difficulty of breathing, constriction of the chest, much palpitation, often accompanied with intermission, weak thready pulse, and syncope. The heart's action occasionally becomes exceedingly slow, or it may be unusually rapid.

The subject of heart affection in relation to Gout is one of considerable interest, more so from the fact that the organ is so frequently affected in acute rheumatism. No conclusive evidence has yet been advanced proving the existence of true gouty inflammation of the heart; the examination of the surface, the lining of the organ, and the valves in gouty subjects, has not shown the presence of deposits; but it must at the same time be allowed, that in cases where the heart has been implicated, they have not been specially sought for. It has been asserted that the post-mortem examination of gouty subjects has frequently revealed the presence of white patches upon the surface of the heart, and Dr. Begbie fancies that these are often the results of gouty inflammation; he is also inclined to think that the endocardial vegetations and puckerings are due to the same cause; no proof, however, has been brought forward in confirmation of these views, and the writer of the present article can state positively that his examinations have failed to detect urate of soda, either in the white patches, the endocardial deposits, or the atheromatous spots. It must also be remembered that the causes which lead to the production of Gout are such as are likely to induce chronic valvular diseases of the heart.

Gout affecting the Respiratory System.—Acute inflammation of the lungs or pleuræ, if it ever occurs from retrocedent Gout, is certainly very rare indeed, although some authors have described these forms of disease; but functional affections of the respiratory organs are of very frequent occurrence in gouty habits, and so probably is a form of bronchitis; the most common manifestations of such disturbance being shown by asthmatic breathing and cough. Occasionally these symptoms are unaffected by ordinary remedies, and yet yield at once to the development of articular Gout, or to the administration of medicines calculated to give relief in ordinary Gout.

Gouty Conditions of the Urinary Organs.—The different parts of the urinary tract appear peculiarly prone to be affected by Gout. The kidneys are undoubtedly implicated in very many instances, and structural alterations are frequently produced, which will be described under the "Morbid Anatomy" of the disease.

From many observations and post-mortem examinations, I am of opinion that gouty inflammation of the structure of the kidney is by no means rare, and that a true deposition of urate of soda takes place as its result. That this may occur early in the gouty life of a patient is certain; possibly it may at times even precede the joint affection. It must be borne in mind, when investigating these cases, that renal calculi are somewhat common in gouty subjects, and the symptoms must therefore be carefully analysed; in irritation of the kidney from a calculus, the pain is more likely to be confined to one side of the loins; albumen is not so frequently present, and if so, it is probably due to a little blood; and, lastly, there is the absence of febrile disturbance.

The bladder and urethra may also become affected with Gout, and a species of chronic cystitis and urethritis induced, especially in old people. Where the irritation of these surfaces is simply the result of this diathesis, the symptoms are greatly relieved or altogether removed when the joints are attacked; but in many instances some organic urinary mischief exists, the symptoms of which are aggravated by the gouty habit.

Gout affecting the Eye and Ear.—Ophthalmia appears to be occasionally of a gouty character, although many of the cases recorded have been the result of purulent urethral affection.

I have recently seen two cases of gouty sclerotitis accompanied with the white deposits of urate of soda on the surface of the tissue.

The occurrence of the little bead-like chalk-stone on the helix of the ear has already been fully described; it has been supposed that the surfaces of the drum and of the ear-bones are at times the seats of like deposits; those I have examined have consisted simply of bone earth, and have not occurred in gouty subjects.

The external ear is at times painfully affected by acute Gout, even to the extent of preventing the patient from resting on it when in bed.

Gouty Affections of the Skin.—If inquiry is made it will be found that skin eruptions are very common in those who are the subjects of Gout, and if they are not absolutely produced by the state of system which leads to the articular affection, still they are evidently kept up by it.

Psoriasis is, perhaps, the most frequent form in which the cutaneous disease manifests itself, and there are records of many cases in which the skin and joint-affection are alternated.

Eczema is likewise not an infrequent accompaniment of the gouty diathesis; sometimes it assumes an acute, sometimes a chronic character.

Prurigo is also met with in connexion with Gout, either in the limited form of prurigo ani, or as a more general affection.

Acne, in the face and other parts, is sometimes found to be closely dependent on the gouty diathesis, and I have known one case in which the patient could predict the advent of a gouty paroxysm from the appearance of these spots.

Diseases occurring in Gouty States of the System.—There are certain diseases to which gouty subjects are especially liable, and amongst these gravel and calculus may be particularly mentioned.

When the pathology of Gout is considered, the occurrence of uric acid gravel cannot be a matter of surprise; from a very early stage deposits of urate of soda take place in the tubules, and Dr. Prout has remarked that occasionally patients void this salt in considerable quantities. Some patients suffer in early life from calculus, in after periods from Gout; in other subjects calculus and Gout alternate, and occasionally the two affections are present at the same time.

Oxalic acid, which is so readily produced from uric acid, not infrequently occurs in the urine of gouty subjects, and may lead to the formation of calculi.

The existence of a gouty diathesis has been regarded as antagonistic to the development of phthisis; I have, however, seen instances in which phthisis and Gout have run a simultaneous course.

Gout and diabetes occasionally occur in the same individual, but the development of the latter is usually followed by the cessation of the former disease.

Condition of the Blood in Acute and Chronic Gout.—The blood undergoes important alterations in Gout, changes which are almost in themselves pathognomonic, and which require to be carefully studied and clearly understood. In the writings of the ancients, nothing definite is found on this subject; and even until the past few years our knowledge of the state of this fluid in Gout may be said to have been of little value.

The blood-corpuscles, as far as yet known, undergo no necessary change either in number or quality; they become lessened in chronic forms of this disease, when the general nutrition of the body is impaired, but not more so than in other maladies. Many of the poorer subjects of chronic Gout, it is true, are pale and anæmic; and among painters and other gouty patients who work in lead, this is often due more to the influence of the metal which has been imbibed than to any other cause.

It is in the serum of the blood that the chief deviation from the healthy standard is discovered; and in this portion it is not so much that the normal constituents are affected, as that excretory substances which should have been eliminated are retained—an effect due to the imperfect action of certain of the excreting organs, more especially the kidneys.

In healthy blood, it is impossible by ordinary tests to discover the presence of uric acid, the quantity being so extremely small—in fact almost inappreciable; but in Gout one can easily not only show its presence, but even obtain it in a crystalline form. It was first proved to be present by the author in 1847.[1] The following process can be adopted for its detection:—The serum of the blood is first dried over a water-bath, then reduced to coarse powder, and treated with hot alcohol; the spirit being removed, the residue is afterwards to be digested for some minutes in distilled water, and raised to the boiling-point; the watery solution is then filtered and evaporated to a thin syrupy consistence. A drop or two of the solution, when heated on a piece of porcelain, with nitric acid and ammonia afterwards added, exhibits at once the murexide test. A small portion of the same solution, if acidulated strongly with acetic acid, and allowed to evaporate spontaneously, gives rise to the crystallization of uric acid, the crystals exhibiting its characteristic form; and, lastly, the syrupy solution, if merely allowed to evaporate without the addition of any acid, exhibits upon its surface, after a few hours, small white tufts of acicular crystals of urate of soda; the nature of the base being determined by the examination of the white alkaline ash left after incineration; the acid by the murexide and other tests.

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[1] Medico-Chirurgical Transactions, 1848.

In the clinical examination of the blood, this process would be too elaborate and tedious; but another method,[1] which answers admirably for practical purposes, is to put about two drachms of the serum in a flat glass dish, somewhat larger than a watch-glass, acidulate slightly with acetic acid, and having placed in the fluid an ultimate fibre from a piece of linen cloth (unwashed huckaback answers well), set it aside in a safe place until the evaporation has proceeded sufficiently far to cause it to become of a gelatinous consistence. If there is uric acid in any abnormal quantity in the serum, the fibre becomes studded with crystals of uric acid, which can be at once recognised by placing the glass under the microscope with a low power, or by the use of a small pocket magnifying glass. I have never yet, after very numerous trials, failed to discover uric acid in the blood of gouty patients by this method, and the test has an especial advantage in only requiring the abstraction of a very small quantity of so important a fluid. It may certainly be stated as a fact, absolutely proved, that the blood in Gout always contains an abnormal amount of uric acid, and that this acid exists as urate of soda. Besides uric acid, urea is frequently found in varying quantities in the blood in this disease, especially when the affection is of long standing, and the kidneys have become much involved and their excreting powers impaired.

In 1849 [2] the writer discovered the presence of oxalic acid in the blood, and since that time has, in several instances, detected it in gouty subjects; its presence appears due to the decomposition of the retained uric acid.

The other alterations in the serum of the blood occasionally met with in cases of chronic Gout, are, the lessening of its specific gravity, due to the loss of albumen, as well as to the impaired nutrition of the body; and the diminution of its alkaline reaction. With the exception of collapsed cholera, and perhaps certain cases of albuminuria, the reaction of the blood is found to be nearer the neutral point in severe forms of chronic gout than in any other disease; this lessening of the alkaline condition probably depends on the deficient action of the kidneys and the retention of acid products, as it is only when the kidneys are much affected that such a state of the blood is observed. The amount of fibrin is always increased when active inflammation of the joints is present, and the augmentation appears to be in the ratio of the inflammatory action; in fact, the fibrin follows the same laws as in other forms of inflammation; hence, in acute Gout, if blood is abstracted, the surface of the clot exhibits a buffed and often a cupped appearance.

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[1] Medico-Chirurgical Transactions, vol. xxxvii. 1854.

[2] Ibid. vol. xxxii. 1849.

Condition of the Blood in the intervals between the Attacks of Gout.—It is a matter of some importance to ascertain the state of the blood in gouty subjects, when no inflammatory action is present; that is, during the complete intervals of the attacks. Although a somewhat difficult task, from the unwillingness of patients to be bled when not suffering from pain or fever, I have been enabled, in a few instances, to collect some facts of importance on this point.

1. In the intervals between the early attacks of Gout no appreciable amount of uric acid was found in the blood.
2. A very marked decrease of uric acid was observed in the blood of patients partially recovered from an acute attack.
3. In chronic Gout the blood, even in the intervals between the exacerbations, was always rich in uric acid.
4. In some cases when symptoms of irregular Gout were manifested, without any accompanying joint disease, uric acid was present in the blood.

State of the Urine in different forms of Gout.—Much error prevails in regard to the alterations which the urine of gouty subjects exhibits during the progress of the disease, and such misunderstandings have partly arisen from the prevalent idea, that the appearance and non-appearance of certain principles in the urine necessarily indicate their presence or absence in the blood; and partly from a want of accurate knowledge of the reaction of healthy urine.

It must be remembered that healthy urine exhibits a strongly-marked acid reaction, and that this is, in all probability, due, not to the presence of a free acid, but to the existence of an acid phosphate, probably the phosphate of soda ($2\ HO,\ NO,\ P_aO_5$), a salt containing two equivalents of water and one of soda to each equivalent of tribasic phosphoric acid. The acidity of urine varies much at different times of the day, and is in close relation to the state of the digestive functions. The fact of the acidity of healthy urine has been specially insisted upon, because the application of litmus papers is not infrequently made both by medical men and patients, and very wrong deductions drawn from the indications thus obtained.

It must be remembered that a strong acid reaction is no proof of the presence of any abnormal state of the urine; and that urate of soda is one of its constituents, a salt which can exist in a solution of the *acid* phosphate without decomposition, and hence the possibility of the co-existence of the urate of soda with an acid condition of the urine.

The amount of uric acid passed by a healthy subject during twenty-four hours is from eight to ten grains—a quantity capable of existing in solution when the urine is in a healthy state.

Having premised thus much, we are better prepared to speak of certain changes which are observed in the urinary secretions in Gout, changes the import of which has been often misunderstood. It has already been proved that the blood in all stages of Gout is invariably rich in uric acid, and, as the kidneys are the only organs which can be shown to eliminate this body, it follows that these organs must be inefficient for their required task when such a condition of the blood ensues. It becomes, then, a matter of some interest to discover whether these organs in gouty cases lose any of their normal power of excreting uric acid, or whether the formation of this acid in the system is increased.

In acute gouty seizures of a sthenic character the urine usually becomes scanty; it is then high-coloured, and not infrequently gives rise, in cooling, to an amorphous deposit, varying in tint from pale yellowish red to dark red, or at times an intense pink. The colour varies under different circumstances, such as the amount of febrile disturbance which is present, the state of the portal system, and that of the biliary secretion also. If a judgment is formed from the inspection of a small specimen, without taking into consideration the limited amount passed in the twenty-four hours, it may readily be supposed that a great excess of uric acid is secreted; and such, in fact, has been the prevalent idea. However, a more careful examination of the urine in these cases leads to a different conclusion; for example: in a set of observations, taking the averages in seven acute cases, the daily secretion of uric acid was found to be under four grains, an amount far below the normal amount, which is from eight to ten grains. The cause of the prevalent idea that in Gout the uric acid exists in the urine in excess has probably arisen from the following circumstances:—During the febrile stage there is usually a deficient flow of urine, the acidity of the fluid is augmented, thus causing the precipitation of the whole of the urates; at the same time the increase of colouring matter in the deposit gives the appearance of a larger amount than actually exists.

It must not be concluded from the above that in any given amount of urine passed in a gouty paroxysm there may not be a larger amount of uric acid than that which is found in health—often it is so; but our remarks apply not to the relative, but to the absolute weight eliminated in the twenty-four hours.

The question whether the deficient excretion of uric acid is due to a loss of renal function, or to a diminished formation of the acid in the system, is at once determined; for it can be demonstrated that at the time that the urine is deficient in this principle, it exists in the blood in abnormal quantities. It is also known that in some other diseases, in which uric acid is formed in augmented quantities, the kidneys are found equal to the task of eliminating such excess, and the blood is thus kept free from any contamination.

The excretion of the urea also is probably somewhat diminished during the febrile excitement of acute Gout, but by no means in the same ratio as the uric acid.

As Gout assumes more and more a chronic character, so it will be observed that the excretion of uric acid becomes gradually lessened, even in the intervals between the attacks; showing that the kidneys are permanently injured, as far as their uric-acid excreting power is concerned; and in the advanced stages of the disease, when the chalk-like deposits are thrown out in various parts of the body, and when the blood is permanently loaded with the acid, the renal organs often lose entirely, or all but entirely, their eliminating property for this principle. The results obtained from a large number of examinations of urine, and in numerous cases of chronic Gout, may be thus summed up:—

The urine is generally paler than in health, lighter in specific gravity, and often passed in augmented quantities.

There is, for the most part, an absence of any deposits on cooling; at times, however, such may occur, especially when an exacerbation of the disease is passing off. The quantity of uric acid eliminated in the twenty-four hours is notably diminished, and not infrequently it is reduced to a mere trace.

The kidneys sometimes throw out uric acid in an intermittent manner; for example, for several days the uric acid gradually decreases, until scarcely a trace is present, then suddenly a large elimination takes place: this has been clearly made out in several cases.

Even in the intervals between the fits, the urine of patients suffering from chronic Gout is deficient in uric acid.

The urea also, in many cases, is slightly diminished, but not in any remarkable degree, if the diet of the patient at the time of examination is taken into account.

A small amount of albumen is very frequently met with, also granular casts; and, in some cases, waxy or fibrinous casts are likewise found.

Secretion from the Skin in Gout.—It is a common opinion that the production of excessive perspiration is the means of getting rid of gouty matter from the system, and the value of hot-air and vapour baths, and other modes of increasing the cutaneous secretion, have been thus accounted for. There are also to be found, in different works, statements to the effect that uric acid is capable of being eliminated by the skin, and that a white, powdery matter is occasionally seen upon the surface of patients suffering from a paroxysm of Gout. I have made many observations upon this subject, which have led me to doubt the accuracy of such statements, and within the last two years have had an opportunity of putting the question to a severe test. A gentleman, suffering from a very decided attack of Gout, went into a Turkish bath, and took precautions to enable him to save a considerable amount of the perspiration which flowed from his body during the operation. To this fluid rectified spirit was at once added, in order to prevent decomposition, and it was afterwards carefully examined by the following process: It was first evaporated to dryness, then heated with absolute alcohol, and the residue afterwards treated with hot water. The alcoholic solution yielded a notable amount of urea, which was obtained as the crystallized nitrate; but no trace of uric acid could be discovered in the watery solution by the most careful search. This observation, coupled with many others above referred to, leads me to believe that the healthy skin does not possess the power of eliminating uric acid, even when it exists in abnormal quantities in the circulating fluid. It must, however, be remembered that the liquid thrown out from blistered surfaces in like cases is rich in uric acid, and it is not improbable that in some cutaneous eruptions, as in the secretion in eczema when occurring in gouty habits, it might be detected. Allusion has been made to a white deposit seen on the surface of patients suffering from Gout: on one or two occasions, when I have had the opportunity of examining such matter, it has been found to consist of epidermic scales, together with dry salts, but to be entirely devoid of uric acid.

Other Secretions in Gout.—It has not been determined whether uric acid is contained in the secretions from the mucous membranes of the bowels in subjects labouring under Gout, and, when evidence of its presence in the blood is beyond doubt, it would be worth seeing if it is contained in the watery excretions produced by the action of saline or other purgatives. In morbid secretions uric acid is often present: thus it is found in the fluid effused in cases of pericarditis, also in ascites, when these diseases occur in subjects in which the blood is contaminated with this principle. It is also found under like circumstances in the fluid effused by the action of blisters, and we can sometimes make use of this fact in diagnosis, employing the blister fluid in lieu of the blood serum. There is, however, one precaution necessary to be observed, which is, to avoid taking the fluid from a part affected with gouty inflammation, as it would appear that inflammation has a tendency to destroy the uric acid in the blood of the part. This fact, if well established, would be of great value in elucidating the pathology of the disease.

The thread experiment may be employed for the detection of uric acid in the blister fluid, as in blood itself.

MORBID ANATOMY OF GOUT.—Gout is seldom fatal, yet opportunities for investigating the morbid appearances produced by the occurrence of the malady, even in its slighter forms, are not difficult to obtain, provided that the gouty history of cases be carefully taken for a considerable period of time in any great public medical institution. It is only by this method that the writer has been enabled to collect the materials of which he now gives a short summary, and which he hopes will go far to elucidate many of the phenomena of the disease.

In one case there had been only two very slight attacks of Gout, affecting the metatarso-phalangeal joint of the right great toe, the first attack occurring two years, the second only one year, before death. The condition of the joint was as follows: On the head of the metatarsal bone was a white patch, made up of minute aggregations of a white deposit, occupying altogether about a tenth of the articulating surface; on the cup-shaped surface of the phalanx, the same sprinkling of white matter was observed upon the cartilage, occupying a greater extent of surface than on the metatarsal bone; on the inner surface of the ligaments, the same substance was here and there observed; the surfaces of the sessamoid bones were free from any deposits, and, although the same joint on the left side, as well as many other small and some large joints were examined, no deviation from the normal state was observed in them. The gouty attacks, it will be remembered, had been confined to one articulation, and this joint only was found to be altered after death.

In another case, only one attack of Gout had occurred, affecting the right great toe very severely, the left but slightly; and similar appearances in the cartilages and ligaments were discovered, but exceedingly slight in the left toe-joint. In this case, also, several other joints were examined, but found to be free from any morbid alteration.

We thus see that even a single attack of Gout leaves marks behind, which appear to be very nearly, if not altogether, indelible; for, in the second case, at least thirteen years had elapsed from the time of the gouty seizure to the death of the patient.

Before proceeding to speak further of the morbid appearances produced by gouty inflammation, it will be necessary to point out the nature of the alteration we have already noticed—that is, the nature and situation of the white deposit. When the alteration in the joint is slight, no appreciable elevation of the surface can be discovered; and if we pass the finger over it, nothing abnormal is detected, nor can we remove the deposit by moderate friction, nor, in fact, by any means short of removing the surface of the cartilage itself. On the other hand, if we immerse the bone for some hours in water at the temperature of the body, or keep it in a cold and weak solution of carbonate of potash, the white matter is slowly dissolved out, and on afterwards drying the bone, the articulating surface appears to be restored to its healthy condition. We can thus show that the deposit is not on the surface of the cartilage, but within it, and that it consists of a material soluble both in warm water and in a weak alkaline solution. A further insight into its nature may be obtained from a microscopical and chemical examination: if, for example, we make a vertical section of the cartilage over the seat of the white deposit, we at once see, either with the naked eye or a simple lens, that the infiltrated matter is most dense near the free surface, and gradually diminishes as it approaches the bone, seldom, indeed, extending halfway into the substance of the cartilage.

If we place the thin vertical section under the microscope, using a quarter-inch objective and low eyepiece, a very beautiful appearance is exhibited; the opaque white matter is observed to consist of very fine crystalline needles or prisms closely interlaced, and according to the density of the network, so is the amount of opacity produced. As the deposit becomes more sparse, many separate crystals are seen, which appear to project into the substance of the healthy cartilage. If we examine horizontal, in lieu of vertical, sections of cartilage, we find that, after we have removed a few slices, the deposit becomes sufficiently thin to allow light to pass freely through it, and the crystals are for the most part seen to be arranged in little clusters radiating from centres, the interspaces being nearly free, or with only scattered crystals. If, instead of using ordinary light, we employ the polariscope, the appearances above described are much intensified, the crystals become strongly illuminated, and more or less coloured; at the same time, the healthy portion of the cartilage, if sufficiently thin sections be made use of, gives a black background.

The chemical nature of the deposit can be readily demonstrated. If slices of the altered cartilage be first washed with a little cold distilled water, to remove any soluble matter, and afterwards digested for some hours in hot water, under 200° Fahr., the infiltrated matter is dissolved, and a solution is obtained which, treated with a little nitric acid and afterwards evaporated in a porcelain capsule, and when nearly dry exposed to the vapour of ammonia, exhibits an intense purple colour from the formation of murexide; on the other hand, if some acid, as acetic, be added to the solution so as strongly to acidulate it, and the whole allowed to evaporate spontaneously to a thin syrupy consistence, crystals of uric acid, more or less coloured, are slowly deposited, and the forms they exhibit under the microscope at once distinguish them; again, if the watery solution, without the addition of any acid, be slowly evaporated to a syrupy consistence, and then allowed to cool, urate of soda, in bundles of crystalline needles, will form on the surface, which can be readily collected, and, if necessary, chemically examined.

If a thin vertical section be obtained from any articular surface which has not been much worn—that is to say, from a joint which has not been often attacked, or is not liable to injury from friction—a distinct organic layer devoid of crystals can be seen by the aid of the microscope, superficial to the deposit, and probably consisting of flattened cartilaginous cells.

As yet we have only spoken of very early cases of Gout, in which merely the ball of the great toe has been attacked; if we take more advanced cases, where the disease has lasted for several years, and in which many of the larger as well as the smaller joints have been implicated, opportunity is afforded of investigating still further the changes effected by the disease.

If a knee has been but slightly attacked, only a few spots or small patches can be detected upon the articulating surfaces of the femur, tibia, and patella; but, if it has been severely affected, a large portion of the surfaces may exhibit the peculiar alteration: the condyles of the femur are often completely incrusted, except at their margin, in the situation of the synovial fringes, the vascularity of which appears to protect this part from the deposit;[1] the concave surface of the tibia is usually less covered; the patella is often extensively coated; and in many instances the fibro-cartilages and crucial ligaments are more or less implicated. In such a knee-joint the synovial fluid is thickened, and in extreme cases contains tufts of urate of soda; the synovial membrane is also seen to be speckled with little white points looking like amorphous granules. When these are placed under the microscope, they are found to consist of acicular crystals radiating from a centre, and forming with polarized light a very beautiful object. In cases of Gout in which the disease has so far advanced as to cause considerable stiffening or complete anchylosis, the ligaments are found to be much infiltrated and thereby rendered rigid; and, as a rule, the movement of a joint is impeded more by the ligamentous than the cartilaginous alteration. Instances are not uncommon in which the movements of the great toe-ball are but partially interfered with, although the ends of the bones are completely covered; but the ligaments in such cases have escaped.

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[1] Dr. W. Budd, Medico-Chirurgical Transactions, 1855.

The shoulders and hip-joints often remain free from disease when most of the other articulations of the body are implicated, but occasionally they present similar appearances to those above described. I have a specimen in which the head of the femur is almost completely incrusted; the ligamentum teres, however, is free, and the movements of the joint are consequently preserved.

The carpus and tarsus are often severely attacked by gouty inflammation, and it is not unusual to find every articulating surface of these bones completely covered with the deposit; also the surfaces of the metacarpal and metatarsal bones, and frequently of several of the phalanges. There is, however, one point of interest in relation to the joints of the great toe, worthy of being borne in mind; it is the fact that, although the metatarso-phalangeal articulation is so constantly affected, the tarso-metatarsal and the phalangeal joints on either side are for the most part free from morbid alteration.

Sometimes urate deposits are found in other articulations. I have met with them even in the arytenoid cartilages, but it requires a special search, not usually made in post-mortem examinations, to discover their presence.

It has been asserted that urate deposit has been seen in bone itself; Cruveilhier found it in the astragalus, os calcis, and patella. Although I have carefully searched for it in bone, I have not yet succeeded in finding evidence of the deposit having originated in this tissue. It is true that deposits lie in contact with bone, as they often originate in the periosteum, and sometimes acquire sufficient size to press on the osseous tissue and cause its absorption. Dr. Charcot, in conjunction with M. Cornil, has recently (1864) published an account of the post-mortem appearances in the case of a female, aged 84, who had suffered from Gout for many years, and had been long crippled and deformed; a full description of the appearances presented to the naked eye is given, as well as those seen in the microscopic examination; in every important respect the results are the same as, and confirmatory of, those above described.

Condition of the Kidneys in Gout.—From the earliest time it would appear that an idea prevailed that Gout and renal diseases were in some way allied; Aretæus held that opinion, as also Sydenham, Morgagni, and others. The idea was derived from the fact that gravel and calculi are so frequently met with in gouty subjects.

In the early stages of Gout, should an opportunity be afforded of examining the kidneys, little or perhaps no alteration will be observed, but occasionally an appearance indicating the action of the disease may be presented to the eye, and the first change usually noticed is found to be due to the occurrence of some crystalline deposits in the organ. In 1849, the writer, when examining the kidneys of a man who had suffered from Gout, but who had died of another disease, found small white streaks which appeared to follow the direction of the tubes of the pyramidal portion of the organ; he also discovered that at the extremity or mamilla of each cone there were certain white points;—Dr. Todd and Mr. Ceeley had previously noticed this fact.

On placing a little piece of the substance of the kidney under the microscope, the above-mentioned white streaks and points were found to be caused by the presence of numerous crystals, prismatic in shape, and consisting of urate of soda, that is, identical with those which form ordinary chalk-stones, and which are found in cartilaginous and ligamentous tissues, but the crystals are usually larger. Shortly afterwards, the same condition of kidney was discovered in numerous other subjects, some of whom had been but slightly affected with Gout, and in one only eight seizures had occurred, and no external deposition or deformity had been produced. At first it was thought that the white streaks were due to the blocking up of the uriniferous tubes, but afterwards the conclusion was arrived at that many of the crystals were imbedded in the structure of the tubes themselves. M. Charcot, in an excellent paper, entitled 'Contributions à l'Étude des Altérations anatomiques de la Goutte, et specialement du Rein et des Articulations chez les Goutteux,' has investigated this subject very thoroughly, and has given good drawings: he comes to the conclusion that, as far as the kidneys of the subject examined by himself were concerned, the deposit, which at first sight appeared to be situated in the intervals or between the tubes, was in reality in part contained within them, and in a great measure amorphous; there were, however, crystals not within the tubes which appeared to radiate from the above matter into the intertubular structure.

It would appear from the observations which have been made both in this country and abroad, that even in the early stages of Gout the kidneys become implicated, probably in some cases much sooner than in others; that deposits of urate of soda take place in the tubes, which afterwards extend into the renal tissues.

CAUSES OF GOUT.—The causes of Gout may be conveniently treated of under two heads: first, those depending on the age, sex, hereditary and other peculiarities of the individual; secondly, those independent of the affected subject.

CAUSES DEPENDENT ON THE INDIVIDUAL.

Hereditary Influence.—Some individuals are undoubtedly more disposed to Gout than others, and in such the disease is liable to be induced by agencies which would be comparatively harmless if applied to other people. There is, in short, a proclivity to Gout which may be inherited; and it is certainly true as regards this malady, that the sins of the fathers are visited upon the children to the third and fourth generation. Instances illustrating the hereditary predisposition to Gout are so frequently met with, that no one who has had the least acquaintance with this disease can have failed to have observed it. My own experience would show that more than half the gouty subjects can distinctly trace their ailment to hereditary taint; and if patients in the upper class of society are exclusively selected, the percentage is found to be considerably greater. There exists a popular idea that Gout frequently skips over a generation, and that it has a peculiar tendency to attack the grandchildren rather than the children: this idea I believe to be erroneous, but at the same time acknowledge that there are often apparent grounds for it. Occasionally the child of a very gouty parent, having the fear of suffering before his eyes, will live such a life as to keep the disease at bay; his children, however, may be fully under the hereditary influence of their grandparent, and liable to a development of the malady from the ordinary exciting causes. When true Gout is met with at an early age, the existence of hereditary taint may be suspected, and in the case of children this cause is certain to be powerfully operative. However, it must not be forgotten that Gout may be acquired at a comparatively early age, by the influence of causes other than that of hereditary predisposition.

Influence of Sex.—Men are much more frequently the subjects of articular gout than women, and the causes of the comparative exemption of females is not difficult to understand, when we are acquainted with the pathology of the disease. As far as hereditary causes are concerned, women are similarly circumstanced with men; but there are many reasons explaining the immunity enjoyed by women. Some of these are intimately connected with the functional peculiarities of the female sex; others depend upon the habits of life more commonly adopted by women. The occurrence of the catamenia during a long period of female life is doubtless a great safeguard against the disease, and, as a rule, whenever Gout does occur in the female, it is only after the cessation of this function. There are, however, striking exceptions now and then met with—instances of the most severe Gout, attended with great crippling and deformity, in comparatively young women; these cases are extremely rare, and most of them capable of explanation.

Influence of Age.—Children are usually free from Gout; and, although I have been assured of some being attacked when very young, yet, in every case where there has been an opportunity of strict investigation into the nature of the affection, there has been good cause to distrust the accuracy of the diagnosis which had been made. Many patients have informed me of their having had Gout when at public schools, and I have no reason to doubt the correctness of their statements, more especially as they have afterwards become the subjects of Gout in its severe forms; I have myself seen the disease fully developed in the great toe at the age of sixteen, but always in youths who have strongly inherited the affection, and who at the same time had not been altogether abstemious. As a rule, the stronger the hereditary predisposition, the earlier Gout develops itself in any individual, and it is rare to find it before the age of thirty, unless some well-marked hereditary predisposition exists.

From a table made by the late Sir C. Scudamore from a large collection of cases it appears that, dividing the period of life from twenty to sixty-five into intervals of five years, by far the greater number of first attacks occur from thirty to thirty-five years of age; below twenty they are exceedingly rare; and after sixty-five they are likewise very few in number. As there appear to be exceptional cases in early life, so also are there in old age, and I have known Gout make its first appearance after the age of eighty, and in one instance when the patient was in her ninetieth year.

Influence of Temperament.—Little that is positive can be asserted in regard to the influence of what is called the temperament of the individual upon the development of Gout. The more acute varieties of Gout are usually found in those of a sanguine temperament and full habit of body; whereas the asthenic and irregular forms occur chiefly in spare subjects of a nervous temperament.

CAUSES INDEPENDENT OF THE INDIVIDUAL.

Alcoholic Beverages.—No one who has paid attention to the clinical study of Gout can doubt the influence of alcoholic drinks, both in laying the foundation of the gouty diathesis, and likewise in exciting attacks of the disease; and, moreover, no one who has carefully analysed the causes of this malady can fail to perceive that different kinds of spirituous liquors differ greatly in their power in this respect. This subject is of so much importance, that we shall not hesitate to dwell shortly upon it. Distilled spirits have certainly less tendency to induce Gout than either wine or malt liquors: the truth of this remark can be proved by investigating the cases which occur in the large cities of England, and, more clearly, by noting the prevalence of the disease in other countries where little of any other spirit is taken. Among the labouring classes of London, Gout is very frequently met with; whereas, among the same class in Edinburgh and Glasgow, it is scarcely ever seen: the former partake largely of porter and beer, the latter almost entirely of whisky, and in no inconsiderable quantities. The same fact is illustrated in the almost entire absence of Gout in many cities on the Continent, especially in Poland and Russia, where a distilled spirit is almost exclusively made use of.

As predisposing to Gout, I cannot from experience say whether all distilled spirits are equally innocuous: brandy, whisky, and gin have certainly little predisposing power; rum has been asserted to cause Gout in the West Indies, but the statement is old, and there is no good clinical evidence in support of it.

In countries where the lighter kinds of wine form the chief or sole alcoholic beverage of large classes of the people, the occurrence of Gout is comparatively rare; as, for example, among the working population of France, most parts of Germany, and, I believe, also of Italy; but it is asserted that in certain parts of Germany, as in Berlin and Munich, where malt liquor is largely consumed, the disease is much more prevalent.

It must not be thought, however, that these light wines can be taken with impunity, for although their gout-producing tendency is small, compared with some of the stronger wines and malt liquors, still it is very decided. Light claret, hock, and moselle are probably the best of light wines.

The stronger wines, as port, madeira, and sherry, probably also marsala, are much more potent as gout-producers, and a free indulgence in their use for several years will very often bring on the disease in those not known to have derived any taint from their ancestors. Port enjoys a very marked reputation in this respect; it is doubtless, as we receive it from Portugal, a wine very likely to cause Gout, perhaps more so than any other. Sherry, however dry and pure, is by no means the innocent beverage, as far as Gout is concerned, that many people imagine; I have met with several cases of severe Gout brought on solely by this wine, and have also known attacks of the disease kept up for an almost indefinite period by the patient continuing the use of it, even in small quantities. Madeira is fully as injurious as sherry to the gouty subject, and doubtless, if freely partaken of for any length of time, would be capable of inducing the malady in those not previously disposed to it. The same remark applies to several other wines, possessing characters closely allied to those of the above-mentioned class.

That malt liquors predispose strongly to the production of Gout is made evident by the frequency of this disease among the labouring classes and artisans of the large cities of England, where porter is so freely indulged in: thus Gout is very common with brewers' men, ballasters, and many others. Even the pale bitter ales, though to many so grateful and useful, will, when too freely partaken of, give rise to the development of Gout, and several cases in which such ale was the sole cause have occurred in my practice.

It only remains for us to investigate the influence of one other alcoholic drink—that is, cider—which forms so favourite a beverage in many of the counties of England, more especially Herefordshire and Devonshire; also, of the United States of America. Concerning its gout-producing powers many and diverse opinions have been held. Dr. Wood of Philadelphia informed me that, as a predisposing cause of Gout, cider cannot be very potent, else the disease would have been more prevalent among the people of New England and the Middle Atlantic States, where it is very commonly drunk. Having taken some pains to investigate the question in this country, the results I have arrived at are as follows:—

1st. That fully fermented cider—that in which the whole of the saccharine matter has been got rid of, and which constitutes genuine rough cider—has but little power in inducing the gouty diathesis.

2d. That sweet and partially fermented cider, when taken in large quantities, predisposes to Gout.

3d. That the latter variety, and even the former, when taken by gouty individuals not much accustomed to its use, is apt to excite an attack of the disease.

Having enumerated the relative powers of different alcoholic liquors in common use in this and other countries, in inducing Gout, it becomes a question of interest to endeavour, if possible, to get some clue as to the causes of such differences; or, if unable to accomplish this, at any rate to point out the known peculiarities of each class of such beverages.

The distilled liquors consist of alcohol, more or less diluted, and combined with very small amounts of volatile oils or ethers: thus, brandy contains œnanthic ether, the peculiar principle of all wines; gin, a little oil of juniper; and so on. They should not hold in solution any non-volatile substances, and should be free from saccharine matter and acidity. The percentage of real or anhydrous alcohol in the different distilled spirits varies greatly, ranging from 70 per cent. in undiluted rum, to about 30 per cent. in gin.

Wines consist of diluted alcohol, combined with certain soluble compounds, as œnanthic and other ethers, free acids and salts, and, besides these, colouring, astringent, and saccharine matters.

The amount of alcohol varies, from about 18 to 22 per cent. in ports, sherries, and madeiras, to 7 to 9 per cent. in clarets and hocks.

The acid and salts consist chiefly of tartaric acid and the acid tartrate of potash; there are also small amounts of other salts, as phosphates of lime, magnesia, and iron.

The amount of alcohol in port wines varies from 17 to 21 per cent.; in madeira it is about the same; in sauterne it varies from 12 to 15 per cent.; in red French wines from 9 to 14 per cent.; in champagne 10 and 11 per cent.; in Rhine wines 6 to 12 per cent., usually from 9 to 10 per cent.

Measured by the amount of acidity, Dr. Bence Jones has arranged wines and spirits as follows:—Sherry, port, madeira, champagne, burgundy, hock, and moselle. The least acid of all alcoholic spirits are geneva and whisky, then rum and brandy, afterwards ale, porter, and stout; all wines are found to be more acid than malt liquors.

Measured by the amount of contained saccharine matter, commencing with the least sweet, spirits, wines, and malt liquors may be thus arranged: geneva, rum, whisky, claret, burgundy, Rhine wines, and moselle have no sugar; then brandy, sherry, madeira, champagne, port wine, cider, porter, stout, malmsey, ale, tokay, samos, paxarete, and cyprus.

The knowledge of the composition of the different kinds of alcoholic fluids has not thrown much light upon their varying powers of inducing Gout, and the following summary includes nearly all that can be clearly made out on the subject.[1]

1. Diluted alcohol, in the form of distilled spirits, has little power in causing Gout, at least in those who are not predisposed to it.

2. Alcohol, when in combination with other substances, as in wines and malt liquors, becomes a potent cause of Gout, and the greater the amount of contained spirits in such beverages, the more powerful their influence in producing the disease.

3. Neither the acid, sugar, nor any known principle contained in these liquors, can as yet be proved to impart to the alcohol its predisposing influence; for wines the least acid, and liquors the least sweet, are found among the most baneful.

4. Alcoholic fluids which have little tendency to cause dyspepsia, and those which more especially act as diuretics, can, as far as Gout is concerned, be taken with greater impunity than beverages of an opposite character.

Influence of the Solid Food in causing Gout.—It is a matter of much difficulty to assign the share that different articles of solid food have in the production of Gout, and likewise to separate the effect of indigestion caused by any article of diet from the secondary influence of the same food after its absorption into the system. Cullen remarked that Gout seldom attacked persons who lived much on vegetable diet, but he added, or persons who were employed in constant bodily labour; and doubtless the disease is rare amongst those who live in the country, working hard, and eating abstemiously. Experience, moreover, has pretty well established this fact—namely, that an excess of food, more especially animal food, favours the production of Gout, and it probably does so by causing an increased formation of nitrogenized compounds, more especially uric acid. Vegetable substances have comparatively little direct influence, unless they cause dyspepsia from their mechanical structure, or other peculiarities. As far as composition is concerned, it is probable that articles of food containing a considerable amount of saline principles, as for instance the salts of potash, are useful in keeping up the activity of the secreting organs, especially the function of the kidneys, and many such substances, even if acid to the stomach, yet tend to alkalinize the blood and urine from the decomposition of the vegetable acid, and the formation of a carbonate of the alkali.

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[1] Nature and Treatment of Gout. Second Edition.

Pie-crust and sweet substances probably are injurious rather from the indigestion they induce than from containing any noxious principles.

Made dishes, and those which are very rich and complex, are liable to upset the stomach, and thus act rather as exciting than predisposing causes of Gout.

Indigestion as a Cause of Gout.—In investigating this subject, great difficulty is experienced in discriminating between the dyspepsia leading to Gout and that which arises from an already formed gouty diathesis. Many persons suffer from dyspepsia all their lives, and yet never exhibit a gouty symptom; on the other hand, some of those most severely afflicted with Gout have scarcely ever felt the sensation of indigestion.

Indigestion, if it causes the production of an increased amount of acidity in the system, can easily be supposed to aid the development of Gout, by causing a less alkaline state of the blood, and hence favouring the insolubility and deposition of the urate of soda in the tissues; it may also act as a predisposing cause by promoting the formation of uric acid itself. The form of dyspepsia which seems most injurious is that which is connected with congestion of the portal system, and increased vascularity of the mucous membranes of the stomach.

Although observations have not shown that exercise has any marked influence upon the amount of uric acid excreted in a given time by a healthy person, still it is certain that want of exercise soon leads to a sluggish performance of the more important functions of the body, and the production of dyspepsia, and indirectly favours the development of Gout.

Influence of Nervous Depression.—Any circumstance which lowers the tone of the nervous system tends very greatly to the development of the gouty paroxysm, although it is questionable if this cause alone can produce the gouty diathesis. The effect of the mind upon the function of the kidneys is well known and easily appreciated, and upon the other secreting organs, although less readily observed, it is doubtless equally potent. Any severe mental labour is often followed by an attack, and so is prolonged sorrow; and venereal excesses probably act by lowering the tone of the nervous system.

Influence of Climate and Season.—There is no doubt that Gout is much less prevalent in hot than in cold or temperate countries, and it would appear from the reports of travellers that the disease is unknown among the natives of the interior of Africa; it is rare, if not unknown, in China, Japan, the East Indies, and Turkey, and much less frequent in the south of Europe than in this country. Great stress, however, must not be laid on these facts, for Gout is scarcely seen among the labouring populations in Poland, Russia, and other cold climates.

The character of the beverages used by these different peoples has probably more influence upon the production of Gout than the climate under which they live.

As far as regards the development of an attack, climate and season have a very decided influence: for example, a gouty man may often escape his accustomed winter attacks by removing from England to Malta or Egypt during the cold season in this country; and the histories of gouty patients afford abundant proof of the comparative frequency of gouty seizures in the winter months.

In the early stages of Gout the attacks are most frequent in spring, and are often confined to that season; after a time an autumnal seizure is added; but when the disease has become further engrafted into the system, the fits may occur at any season, and at most irregular intervals.

There are occasionally found exceptions to the above rules; for some patients, owing probably to individual peculiarities, suffer far more in summer than in winter.

In warm climates and during hot seasons the function of the skin becomes more active, and this fact affords an explanation of any influence dependent on these circumstances.

Influence of Lead Impregnation in causing Gout.—About fifteen years since I was struck with the fact that a large percentage of the gouty patients who had come under my care in hospital practice consisted of painters, plumbers, or other workers in lead, and a more careful investigation of the subject forced the conclusion on my mind, that the influence of this form of metallic impregnation in inducing a gouty condition of the system was very considerable.[1] Since that period I have made many observations on the point, and the conclusions may be summed up in a few words:—

1. Among the patients in London hospitals, a very large proportion of the gouty (about 30 per cent. in my hospital practice) have been subjected to the influence of lead: many of these have had painters' (lead) colic; some have suffered from wrist-drop or more severe forms of lead paralysis; and all have exhibited the peculiarly characteristic blue line on the gums.

On careful inquiry into the habits of these men, nothing remarkable has been elicited; they have been about as temperate as other men employed in different occupations. It is not painters alone who form this high percentage, but plumbers, composition-doll makers, workers in lead mills, and others whose trades have caused them to be exposed to the use of lead. It may be considered as established, that the metal lead acts as a powerful predisposing cause of Gout.

2. Many cases have occurred which have induced me to believe that individuals suffering from the gouty diathesis are more susceptible of the influence of lead than the majority of other people. It is a well-known fact, that when the drinking water in any house is slightly impregnated with this metal from the cistern or pipes with

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[1] Transactions of Medico-Chirurgical Society, vol. xxxvi. 1854.

which it has come in contact, some of the residents in that house may experience the symptoms of saturnine poisoning, while the rest may be unaffected, although drinking an equally large quantity of such water; thus proving that some people are peculiarly susceptible to its influence: some remarkable examples of this fact have come under my notice. In the medicinal administration of lead preparations, as in cases of hæmorrhage, or excessive mucous discharges, it will be observed, if patients be closely watched, that the blue line appears on the gums in some cases with extreme rapidity, and that even colic pains are soon experienced; but that in others the use of the lead salts can be continued for a lengthened period, without the production of any such phenomena. In several instances where patients have proved to be very easily affected, it has been found that they were of a gouty habit, and many of them had repeatedly suffered from severe attacks of the disease.

3. In some instances, severe attacks of Gout have been induced in gouty patients by the medicinal administration of lead salts, on account of the occurrence of epistaxis or other forms of hæmorrhage; and the frequent recurrence of the seizures, whenever the medicine has been repeated, has satisfactorily shown that the phenomena were to each other in the relation of cause and effect.

4. On investigating the physiological action of the administration of lead salts, it is discovered that a very decided effect is produced by that metal upon the secreting power of the kidney, as far as uric acid is concerned; the function is notably diminished.

5. The blood of individuals suffering from lead paralysis always contains an abnormal amount of uric acid; and the same, probably, holds good in all cases of lead colic.

6. There are many facts which seem to show that the influence of lead, when uncombined with that of fermented liquor, is scarcely able to produce Gout; for although in England Gout is so common among those who work with lead preparations, yet neither in France nor Scotland has the connexion been noticed. In Edinburgh, however, even lead poisoning appears to be much less frequent among painters than in England.

Before concluding this subject, it should be observed that it has long been a matter of medical observation, that lead poisoning often gives rise to pains in the limbs which have generally been regarded as rheumatic or neuralgic in character. These pains, however, which appear to be likewise produced by some other metals, must not be confounded with Gout, with which they probably have no relation.

PATHOLOGY OF GOUT.—Our space is insufficient to allow of even a brief summary of the different views which have been held from time to time, and by different writers, of the real nature of Gout. As the disease has been known from remote antiquity, and as it is one which has always appeared to interest mankind, it is not to be wondered at that very numerous and diverse views have been advocated. The existence of an altered condition of the blood has been always a popular belief among the ancient as well as modern pathologists; but various opinions as to the nature of the alterations have been held. The ancients, as was their wont, ascribed Gout to the superabundance of phlegm, bile, and other natural secretions in the system, and they attributed chalk-stones to the concretions of such matters in the affected parts; and such ideas were held by some physicians even up to a comparatively late date. There have not, however, been wanting supporters of a totally different doctrine—men who, discarding all ideas of a morbid state of the fluids of the body, have looked upon the disease as dependent upon an alteration in the structure or functions of the nervous, vascular, and other systems.

Cullen was the great supporter of the anti-humoral doctrine, and brought forward many cogent reasons for not regarding Gout as due to an altered condition of the blood, or the secretions therefrom. Cullen was a physician whose views are always worthy of consideration, and although many of his statements can now be shown to be erroneous, still a short summary of his objections may not be disadvantageously given, if only for the purpose of being combated. Cullen thought that there was no evidence of the presence of any morbid matter in gouty persons; that neither the blood nor secretions from it had been proved to be altered in this disease, and that previous to an attack there were no symptoms indicating such a change; but that the balance of evidence was against this idea, seeing that many individuals before a seizure were apparently in unusually good health. From what has been stated already in the present article, it will not be difficult to show the error of the above opinion; for it has been demonstrated beyond doubt, by clinical evidence, that the blood is invariably altered in Gout, from the presence of uric acid in the form of urate of soda; that the secretions from the blood are likewise influenced; that symptoms are usually present before the development of the articular inflammation; and, lastly, that the inflamed parts are always altered by the secretion into their structures of the same urate of soda—a phenomenon which has never been shown to occur except in this disease, and which may therefore be regarded as pathognomonic.

Cullen, who was necessarily aware of the occasional presence of chalk-stones in gouty subjects, overcame the difficulty by stating that such deposits only occurred now and then, and after the disease had been present a long time; and, in fact, regarded chalk-stones as accidental phenomena, and in no way essentially connected with the disease. Cullen considered Gout to depend upon a peculiar conformation of some portion of the animal economy, more especially the nervous system; he regarded the chief exciting causes, such as intemperance, indigestion, cold, and other depressing influences, as acting upon the nervous centres, and looked upon most of the symptoms of retrocedent Gout as affections of the same kind.

Cullen's theory of Gout is best expressed in his own words:—"In some persons there is a certain vigorous and plethoric state of the system, which, at a certain point of life, is liable to a loss of tone in the extremities. This is, in some measure, communicated to the whole system, but appears more especially in the functions of the stomach. When this loss of tone occurs, while the energy of the brain still retains its vigour, the *vis medicatrix naturæ* is excited to restore the tone of the parts, and accomplishes it by exciting an inflammatory affection in some parts of the extremities. When this has subsisted for some days, the tone of the extremities and of the whole system is restored, and the patient returns to his ordinary state of health. This is the course of things in the ordinary form of the disease, which we name *regular* Gout; but there are circumstances of the body, in which this course is interrupted or varied. Thus, when the atony has taken place, if the reaction do not succeed, the atony continues in the stomach, or perhaps in other internal parts, and produces that state which we have, for reasons now obvious, named *atonic* Gout.

"A second case of variation in the course of the Gout is when, to the atony, the reaction and inflammation have, to a certain degree, succeeded; but, from causes either internal or external, the tone of the extremities, and perhaps of the whole system, is weakened; so that the inflammatory state, before it had either proceeded to the degree, or continued for the time, requisite for restoring the tone of the system, suddenly and entirely ceases. Hence the stomach and other internal parts relapse into the state of atony, and perhaps have thus increased by the atony communicated from the extremities; all which appears in what we have termed *retrocedent* Gout.

"A third case of variation from the ordinary course of Gout is when, to the atony usually preceding, an inflammatory reaction fully succeeds, but its usual determination to the joints is by some circumstances prevented; and is, therefore, directed to an internal part, where it produces an inflammatory affection, and that state of things which we have named the *misplaced* Gout."

It is as easy to show the fallacies in this theory of Cullen concerning the nature of Gout, as to refute the many dogmas which he puts forward; and, therefore, the mere enumeration of it must be considered sufficient. Since Cullen's time there have been writers on this disease, who, even with the knowledge of some at least of the facts recently discovered, still cannot bring themselves to admit the truth of the humoral doctrine of Gout. The late Sir C. Scudamore, although confessing many difficulties, was, up to a few years before his death, of opinion that there existed a species of plethora. The subject of chalk-stones was already a difficulty with him, and the discovery of the constant presence of uric acid in the blood of gouty patients shook his confidence in his old ideas upon the subject. A recent writer, Dr. Gairdner, believes in the existence of a state of plethora of the chylopoietic organs as a constant accompaniment of

Gout, and thinks likewise that the veins of the part are in a varicose state, that the heart is oppressed with a flood of returning venous blood, made impure from the non-elimination of urea, uric acid, and biliary matters; and he thus views the phenomena of a paroxysm of Gout:—"Venous congestion I consider the first condition essential to the formation of the gouty diathesis. It is no new observation; it is found interspersed through the writings of all former authors. Even those who adopt explanations inconsistent with such a state of things, notwithstanding admit it. This state of the blood was first clearly announced as the great cause of Gout by Galen, whose opinions have continued to influence the minds of succeeding physicians in a greater or less degree to the present day. The truth of the fact being, I imagine, unquestionable, it will always continue to embarrass the doctrines of those who advocate opinions with which it is incompatible. But the great venous canals of the body, as well as the larger arterial vessels, are endowed with a resiliency which enables them to struggle well against the flood of returning blood. This fluid, then, is compressed between two opposing forces—that, namely, which is derived from the heart and arterial system, urging it forward on its course, and, on the other hand, the antagonistic resistance of the great veins leading to the right auricle. Under this compression, I believe that the vessels give way, and a true hæmorrhage is occasioned in the part affected. If the rupture takes place in a minute capillary, carrying the serous portion of the blood only, œdema is the consequence; but if the burst bloodvessel be one carrying red blood, a true ecchymosis is formed."

And again:—"It will surely be admitted that the capillary and nutrient vessels, distributed on the extreme and sentient fibrillæ of the nerves, are affected in the same manner as the larger venous trunks. I believe these distended capillary vessels are the real seat and cause of the painful phenomena of Gout. Is it not credible that such vessels, dilated so as to admit fluids for which they were not intended, and bound down by the firm fasciæ, in which Gout has its usual seat, may give rise to much suffering?"

The great objection to Dr. Gairdner's views is, that there is no proof of the occurrence of hæmorrhage during the gouty seizure; no one has seen the ecchymosis spoken of by him as a constant attendant on it, whereas other and frequent changes are invariably observed in the inflamed part. It is, indeed, true that abdominal plethora is often present in gouty habits, especially when the disease has been induced by high living; but on the other hand, in many cases, even when the disease is of an inveterate character, no symptoms indicating such a condition are discoverable. Furthermore, congestion of the chylopoietic organs, accompanied with obstructed cardiac circulation, is very frequent and of long duration, and yet no Gout is developed.

Many other authors have, within the last half-century, published and maintained views as to the nature of Gout; amongst whom, in this country, may be mentioned Murray, Forbes, Parkinson, Wollaston, Parry, Sutton, Sir E. Home, and Sir Henry Holland. The reflections of the last-named physician on the subject are well worthy of attention. In France the principal authors have been Cruveilhier, C. Petit, Barthez, Guibert, and Trousseau. Some of these have been inclined to favour the views of the solidists, but perhaps the opinions of the majority have had a decided tendency to humoralism.

After these short expositions of some of the principal opinions which have been held of the nature of Gout in recent times, and which have proved to be wholly or in part fallacious, it is necessary to lay before our readers the view which we think accords more completely with facts than any other which has been proposed, and one which will explain nearly all the phenomena presented by the disease. In the first place, "it is essential to the production of this form of articular inflammation that the blood should contain an abnormal amount of uric acid, or rather of urate of soda; and even the phenomena which constitute irregular Gout, or are regarded as gouty manifestations, demand for their occurrence the presence of the same salt in the circulating fluid."

The truth of this proposition has been proved by some hundred clinical observations upon cases of articular inflammation; and although it has not been frequently demonstrated in cases of irregular Gout, yet this has been done sufficiently often to make it matter of certainty. It will, therefore, be unnecessary to bring forward any further evidence of the correctness of the proposition.

It must not, however, be supposed that an excess of urate of soda in the blood constitutes Gout; this would be erroneous, for the salt is occasionally present in large quantities, and yet no gouty phenomena are manifested; but the individual so circumstanced may be looked upon as especially prone to its development, if other circumstances arise which favour its production.

In the next place, "gouty inflammation is invariably accompanied with the presence of urate of soda in the inflamed tissue."

We have already given much evidence of this fact, and in no case in which real gouty inflammation has been shown to have occurred have the morbid appearances failed to present themselves when sought for.

Furthermore, "it can be shown that the amount of deposited urate of soda is not in proportion to the intensity of the inflammation, and that in some structures the infiltration may ensue and scarcely give rise to any inflammatory action; facts tending to the supposition that the deposited matter may be looked upon as the cause rather than the effect of such inflammation."

It is most important, as bearing considerably upon the true pathology of Gout, that the above proposition should be fully substantiated, and it is not difficult to bring forward much evidence in its favour. If an opportunity occurs of examining a joint, as the knee, which has been but once attacked, no great amount of alteration may be exhibited, although the inflammation has been intensely acute: on the other hand, after a joint has become, as it were, callous, considerable deposition can be shown to occur without the production of much local inflammation. The same fact is better illustrated in the case of the ear, in which, as before stated, urate of soda is frequently deposited in the fibro-cartilaginous tissue. This phenomenon, although at times recognised from the sensation of heat, pricking, and tenderness of the part, yet more commonly takes place without the production of any symptom—the patient being, in fact, quite unconscious of its occurrence. In many chronic forms of Gout it is not unusual to find large collections of the white matter formed near the surface, with comparatively little constitutional disturbance, and any one watching the progress of such a case must soon become convinced that the elimination of the salt is not an effect of the inflammation. To show that it is probably the cause is equally easy: let us compare, for example, the articulating surfaces of the knee-joint with the fibro-cartilage of the external ear, in respect to their liability of becoming inflamed from the presence of foreign bodies or the infliction of injuries; irritation of the former is known to be most serious in its results, from the acute action which is set up, whereas considerable damage may be done to the latter without any but the slightest inflammation ensuing. Would not a like difference exist in the same tissues if a substance foreign to their constitution became infiltrated by the action of disease?

"The inflammation of the gouty paroxysm tends to the destruction of the urate of soda in the blood of the inflamed part, and probably also of the salt which has been thrown out."

When describing the blood and secretions of gouty subjects, allusion was made to the fact that, although the fluid effused from the action of a blister usually contains uric acid, yet an exception occurs in the case of its being applied over an acutely inflamed surface. Under these circumstances, it would appear that the uric acid is destroyed by the presence of the inflammatory action; and, if this be true, the gouty paroxysm is, at least to some extent, a salutary process, tending to rid the system of accumulated uric acid; but, as the fit is always accompanied by local mischief, the good effected is by no means unalloyed.

"Gouty deposits do not take place indiscriminately in any situation or in any tissue; but a selection is made, in close relation to the vascularity of the part."

If the remarks on the morbid anatomy of Gout be referred to, it will be observed that the structures most liable to become affected are those possessing little vascularity; as, for example, cartilage, fibro-cartilage, ligament, tendon, and synovial membrane. It will be seen, also, that the deposit, as it were, avoids the contiguity of bloodvessels as exemplified in the knee-joints, in which the surfaces in contact with the synovial fringes are free. Again, in the cartilage itself, the deposition, although interstitial, commences near the free surface, and gradually penetrates deeper into the tissues, but, even in extreme cases, scarcely extends beyond a third of its thickness, and is always at a considerable distance from the bloodvessels of the bone.

It is probably owing to this freedom from deposition which vascular tissues enjoy, or to the rapid destruction of the urate of soda when placed under circumstances which bring it into contact with bloodvessels, that acute gouty inflammation does not affect the covering or lining membranes of the heart, as is the case with rheumatic inflammation. The morbid changes which are often found in the valves of the heart, or the lining membrane of the aorta, form no exception to this statement, for they are not due to the presence of urate of soda, but to an alteration of an entirely different character.

"The kidneys are often, if not always, implicated in Gout, and the affection, possibly only functional at first, soon becomes structural. The urinary secretion is likewise altered."

Under the "Morbid Anatomy" of the disease it has been affirmed that, in all cases where Gout has existed for any length of time, some alteration is found in the kidneys; deposition within or external to the tubuli uriniferi is discovered, and the normal structure of the secreting apparatus is injured. In chronic cases the peculiar shrivelled or gouty kidney is frequently met with. As yet I have never seen this latter form of kidney disease without finding the white deposition, and I cannot help thinking it probable that the presence of the urate may be the exciting cause of the subsequent changes which ensue in the structure of the kidney.

The kidney affection, whether functional or organic, readily explains the altered state of the urinary secretion:—The functional disturbance may cause the defect and irregularity of the secretion of uric acid; the organic alteration accounts for the further diminution in the excretory power of the organ, and the frequent presence of a small amount of albumen in the urine.

"The impure state of the blood, due to the presence of urate of soda, is probably the cause of the disturbance which often precedes the gouty paroxysm; that is, of the so-called premonitory symptoms, as well as most of the anomalous affections (irregular Gout) to which such patients are liable."

That suppression of the whole urinary secretion, such as occurs in intense renal congestion and advanced forms of albuminuria, gives rise to many and alarming symptoms, is a well-known and acknowledged fact; and hence it is reasonable to suppose that a suppressed excretion of one of its constituents should manifest itself by symptoms of a less intense and fatal character. Possibly some of the symptoms may be due to the reaction consequent upon an attempt at deposition of urate of soda in certain unusual situations. Dyspepsia, for example, is very common in albuminuria; it is also frequent in persons of a gouty diathesis.

"The causes which predispose to Gout, independent of those connected with individual peculiarity, are either such as produce an increased formation of uric acid or which lead to its retention in the blood."

Although our knowledge of the causes which lead to the undue formation of uric acid is most imperfect, yet there can be little doubt that over-feeding, especially in regard of animal food, portal congestion, and deficient exercise, aid very much in the production of its excess; and it is known from clinical experience that these are predisposing causes of Gout, as appear to be also lead impregnation, the use of malt liquors, wines, and so on, which probably lead to a defective elimination of the same acid. Deficient nervous energy, arising from mental or other causes, seems to act in the same manner.

"The causes exciting a gouty fit are those which induce a less alkaline condition of the blood, or which greatly augment for the time the formation of uric acid, or such as temporarily check the eliminating powers of the kidneys."

The deposition of urate of soda is caused by its insolubility, and this may arise either from the large amount which is formed, or from the serum of the blood becoming less capable of holding it in solution. The blood serum is alkaline in reaction, and this condition may become lessened from various causes, especially from deficient action of the skin, the taking of a large amount of acid into the stomach, and, perhaps, an increased formation of some acid, arising from dyspepsia: all the above-named circumstances will, it is known, often excite a gouty attack. It seems probable that there may be at times a great temporary increase of uric acid found in the system, by causes giving rise to an attack of dyspepsia.

"Deposits of urate of soda in the textures of the body never occur but in true Gout."

It has already been shown that urate of soda invariably accompanies gouty inflammation, and it can be equally proved that it is not thrown out under other circumstances. There are, indeed, many statements which appear at first to throw doubt upon the correctness of the above proposition, but which upon closer examination can be shown to be erroneous. I have seen white nodules on the ears of young people who have apparently had no gouty tendency: these have been found to contain fat and amorphous granular matter, but no crystals of urate of soda. I once was shown a large tumour, taken from the scalp of a young woman, and was assured that it had been analysed and found to consist of urate of soda, and that there certainly was no gouty tendency in the patient. On examining a portion of the tumour, no trace of uric acid could be discovered in it.

Diseased joints from rheumatoid arthritis, and other chronic diseases, have been also asserted to be covered with a urate deposit: in all such cases I have shown that bone-earth concretion has been mistaken for urate of soda.

In examining the great-toe joints of a large number of bodies, a little white spot was seen in two instances; yet no Gout had been known to have occurred during life. One of the individuals had been a cabman, and had granular kidneys—he had died from an injury; the other had died from delirium tremens. In each case one foot only was affected, and the space covered with the deposit did not exceed a sixteenth of a square inch. These spots were doubtless indicative of very slight gouty inflammation, and in the investigation of cases of confirmed Gout it is extremely common to find that patients have complained of twinges and slight tenderness of the great toe for several years before the occurrence of a distinct and unmistakeable paroxysm.

Explanation of other Phenomena which occur in Gout.—There are certain peculiarities in the history of Gout which still require explanation, one of which is the fact that gouty inflammation in its first visitation generally attacks the ball of the great toe. Boerhaave and Van Swieten tried to explain this by supposing that Gout chiefly attacked those tissues in which the fluids have most difficulty in passing through, as the periosteum, tendons, nerves, membranes, and ligaments, and such as are most remote from the heart, most pressed upon and injured, and most subject to cold and moisture. I believe that there is much truth in these remarks, although expressed in terms which are not conformable to the pathology of the present day. Let us endeavour now to explain the phenomenon.

The great toe contains a considerable amount of tissues peculiarly liable to become the seat of the deposition of urate of soda; as, for example, the cartilages and ligaments, tissues having either little vascularity or nourished independently of bloodvessels: the great toe being very remote from the heart, the circulation is weaker there than in many other parts, weaker than in the hips or knees. These remarks, however, both with regard to the tissues and the distance from the heart, apply even with greater force to the phalangeal joint of the great toe than to the metatarso-phalangeal joint, and apply also to the joints of the smaller toes; but, on the other hand, this latter joint is more subject to injury by pressure; it often has to bear the whole weight of the body, and sudden shocks—as, for instance, from false steps—are first felt in this articulation. In cases where the great toe has not been attacked, some peculiarity has been present in the conformation of the foot, which has had the effect of throwing the pressure on some other part.

That the metatarso-phalangeal joint is liable to injury, I have been able to ascertain from the examination of several great-toe joints in subjects who had never had Gout; and it was found that in six instances only out of twenty were these joints absolutely healthy, there being in the rest more or less evidence of ulceration of the cartilages. The reasons for the great toe of one side of the body being affected apply equally to the other; and hence the disease not uncommonly attacks first one and then the other, within the space of a few hours or days.

It is not difficult even to explain the sudden shifting of the inflammation from one joint to another: it must be remembered, that the deposition precedes the inflammation, and it is well known that the establishment of inflammation in one part is often followed by its subsidence in another. The nervous connexion through the spine may also explain the alternation of inflammation so often observed in symmetrical joints.

In explanation of the reason why numerous joints are attacked as Gout gains ground, or becomes more engrafted into the system, it may be advanced that the cartilages and ligamentous structures of the earlier implicated articulations being infiltrated with the urate, and the blood still remaining impure from the presence of the salt, other surfaces are required to be selected. The defective circulation in the external ear, from the nature of its structure and its exposed situation, is probably the reason why the small urate nodules are so frequently found upon it. I may remark that, up to the present time, I have never seen the concretions on the ears of females; this immunity may arise from their being usually covered: individuals with cold ears seem to be most frequently affected with them. The cartilaginous, fibrous, and ligamentous tissues are peculiarly susceptible of becoming the seat of the deposit, partly from their little vascularity, and probably also from the fluids in these structures being less alkaline in reaction than the blood itself, and liable to become neutral or even acid. After death, in chronic gouty cases, the synovial fluid has been found in a few instances distinctly acid.

The explanation of the comparative immunity from Gout enjoyed by females is to be sought for in their freedom from the influences of many extraneous causes, and their possession of a function which has a tendency to rid the system periodically of superfluous blood. As a rule, women take much less wine and beer than men, and altogether lead lives of greater prudence. After the cessation of the catamenia, women become more liable to gouty paroxysms. Women who inherit Gout strongly, even if they live very carefully, are apt to suffer from the irregular manifestations of the disease; and the same remark applies to men who, inheriting the disease, and having the fear of it before their eyes, have from early life studiously avoided the causes which engender it. The reason of the almost certain recurrence of Gout, unless the greatest care be taken to overcome the tendency, must be sought for in the fact that the causes of the increased formation of uric acid in the system, and of its defective elimination, are generally irremoveable; the periodicity of the disease may be due to the gradually increasing impurity of the blood from the time of the purification which occurs during the fit, and also to periodicity of the exciting causes—as the recurrence of the vernal and autumnal changes.

DIAGNOSIS OF GOUT.—To make a correct diagnosis in cases of joint disease is a matter of importance, not only as regards the treatment, but as respects the prognosis: it is likewise often one of great difficulty, and always requires great care, even with those who have had most experience in the subject.

To determine if a case be of a true gouty character or not:—

The history of the case should be fully inquired into; it must be remembered that Gout is strongly hereditary, and therefore, if either parent or grandparent of the patient suffered from it, the probability of his joint affection having the nature of Gout is much strengthened.

The age should be taken into account; Gout is very rare before puberty, not common till after thirty-eight or forty years of age; it may occur at a very advanced age.

The sex of the patient influences the diagnosis; Gout is much more frequent in males than females; in the latter it is seldom seen till after the catamenia have ceased.

The mode of life of the patient for the several past years should be taken into consideration. Wine, malt liquors, and much animal food, tend to produce Gout; spirits have little effect. The history of the disease in its early stages should be inquired into. If we discover that the ball of the great toe was first and specially affected, and that the intervals between the attacks were of considerable duration, the conclusion that it is true Gout is almost certain to be correct; but if the history has not been of this characteristic nature, it must not be concluded that the affection is not gouty. An error may sometimes arise from laying too much stress upon the toe affection, to the exclusion of other symptoms; I have seen a great toe swollen, tense, red, and hot, and having every appearance of being attacked with intense gouty inflammation, which has afterwards been proved to depend on pyæmia. In this instance, a day or two from the commencement of the seizure, other parts were implicated, and the presence of pus was manifest; from the very first the amount of constitutional disturbance was far beyond that which occurs in Gout affecting one small articulation.

The character of the symptoms should not be neglected. It must be remembered, that in Gout the pain is generally severe; during the early stage of the inflammation the joint is very tense; that it subsequently pits, or is œdematous, and lastly desquamates; that the febrile disturbance is usually moderate, and in proportion to the extent of the local inflammation. The presence or absence of periodicity in the attacks must not be overlooked; Gout, especially for the first few years, is almost invariably periodic, complete and long intervals occurring between the paroxysms.

In true Gout, acute inflammation of the heart does not occur; in rheumatism cardiac complication is frequent.

If a deposit of urate of soda can be discovered either in the external ear of the patient, in the tips of the fingers, the bursæ over the olecranon, or in any other situation, it is a matter of certainty that the patient has the gouty diathesis. Great care, however, must be taken not to confound enlargements of other kinds, as of the ends of the phalanges, or simple bursal swellings, with those produced by the deposition of urate of soda.

An examination of the blood of the patient almost decides the point: this can be effected if only a single ounce is drawn from a vein, as not more than one or two drachms of the serum are required for the thread experiment. The fluid from a blister may be used instead of blood serum, although a negative result obtained from it is not so satisfactory.

Lastly, the presence or absence of a trace of albumen in the urine, if the case be of a chronic character, may afford some assistance, as this symptom is very frequent in Gout, and dependent on the slight kidney affection which so commonly ensues after the disease has lingered in the system for a few years.

A case, showing the importance of attending to the above differential points, has within the last few weeks come under observation.

A woman, aged 35, married, with one child, has for many years worked in a laundry, and has been necessarily exposed to damp, and great alternations of temperature; says she has always been temperate, but has drunk beer, and now and then a little spirits.

The patient is very deaf, which renders it difficult to ascertain every point either in her previous history, or in that of the disease, and which at first obscured the diagnosis. About two years since she had swelling of the left knee, and thinks this joint was alone affected; she was unable to move about for five or six months. About a year from the commencement of the knee affection the feet and ankles were attacked and the great toe was implicated; does not remember whether it was the metatarso-phalangeal or the phalangeal joint; the attack lasted a month or so. From this time to the present has been frequently obliged to keep her bed from joint disease, both the upper and lower extremities being involved. When first seen, both knees were tender and swollen, as likewise the ankles; some tenderness also of two or three of the phalangeal joints of the hands, and the first phalanx of the left index, and the same joint of the right middle finger; these are considerably thickened and swollen, and their mobility much impaired; no visible deposits either in ears or elsewhere. No amount of febrile disturbance present. Catamenia regular. Urine free from albumen. None of her relations had suffered from joint disease.

There was considerable difficulty in arriving at a safe conclusion in this case.

Against the disease being Gout, and in favour of its being rheumatoid arthritis, there was the following evidence: the sex of the patient, her somewhat early age, and the catamenia being still present; the supposed moderate abstinence from malt liquors (though she always took three pints of beer a day, with some spirits); the absence of any affection of the great toe in the first attack, and the question as to which joint of the toe was implicated in subsequent seizures; the duration of the disease and its almost progressive character from the first; the non-discovery of chalk-like deposits in the body; and, lastly, the probability of the joint affection being brought on by cold.

On the contrary, in favour of its being Gout were the following circumstances: the appearance of general good health; the distinct interval of six months between the first and second seizures; and the probability of the patient taking more malt liquors than she herself allowed.

To remove any doubt upon the subject, a very small venesection was performed, and upon analysis the serum was found to yield a large quantity of uric acid by the thread experiment. This was almost decisive of its being Gout; but on very close inquiry, finding that the elbows had been affected several times, the state of the bursæ over the olecranon process was examined, and in the right bursa some thickening was detected, as likewise the presence of two or three little flattened masses, doubtless of urate of soda. The presence of these little masses in the bursa, conjoined with the fact of the blood being rich in uric acid, fully established the nature of the case; and this discovery was of no small importance, not only in the treatment of the attack, but as to the means to be taken in future to keep the disease from making further inroads in the system.

Prognosis of Gout.—An attack of acute articular Gout is probably never fatal, and individuals are often seen who have suffered from severe paroxysms for many years, and yet appear to have experienced little or no injury beyond their sufferings at the time. If the intervals between the seizures continue to be of fair duration, as one year or half a year, when the patient is beyond middle age, the prognosis is favourable, and there is no reason why any appreciable shortening of his life should ensue, provided he is willing to live according to rule, and is not exposed to accidents or other powerful causes of the disease. In confirmation of this statement is the fact that robust-looking persons of very advanced age are not infrequently seen, who have been the subject of periodic visitations of Gout for a great number of years. When, however, in comparatively early life, the attacks are frequent and prolonged, the prognosis becomes much less favourable, and especially if the urine exhibits any trace of albumen, either during the paroxysms or in the intervals of freedom from them.

The appearance of the patient's urine helps us in making a prognosis. If it was formerly turbid from urates, or if it gave rise to a deposit of crystallized uric acid, and has become of late clear and of a paler colour, the change probably indicates that the kidneys have to a considerable extent lost their power of eliminating uric acid, and that which seems to the patient a favourable change is in reality a sign of a serious structural alteration in an important secreting organ.

It is a grave sign in Gout to find the urine pale, the specific gravity exceedingly low, and the fluid devoid of uric acid; and if, in addition to this condition, albumen is likewise present, the indication becomes still more unfavourable.

Chronic Gout has a decided tendency to shorten life, and this fact is recognised by insurance companies, who, however, do not appear to make much distinction between the acute and chronic forms of the disease: at different offices varying rates are adopted.

In the early attacks a patient is likely to inquire of his physician, if it is possible to prevent a return of his ailment—a question tantamount to asking if there is any known method of absolutely eradicating the tendency to Gout from the system.

There are records of individuals who have experienced but one attack, though they have lived to a great age. I have known thirty-five years elapse between a regular attack of Gout in the great toe and the patient's death, which took place after he had attained his seventieth year. Several cases have come under my observation in which the disease, after having recurred periodically for many years, gradually declined in intensity and duration, and at last altogether disappeared.

The appearance of Gout can never be looked upon as a good omen; a statement contrary to a once popular opinion.

The greater the age at which Gout first seizes the individual, the more satisfactory the prognosis.

If it attacks very young subjects, the future prospects are bad.

Hereditary is generally much less tractable than acquired Gout.

The appearance of chalk-stones on the surface is always inauspicious, even if confined to the helix of the ears.

Gouty patients are more liable to suffer severely from accidents and exposure than the majority of people; the more the kidneys are implicated, the less able are they to withstand the effects of shocks upon the system.

In concluding the subject of the prognosis of Gout, I will remark, that I consider that a single fit of Gout, however slight, should be looked upon as an intimation that the patient cannot go on with impunity in his then habits of life; it is a warning that either he must change them or expect returns of the disease, which, as time advances, are certain to increase both in frequency and duration, and both embitter and shorten existence.

On the other hand, I am equally persuaded that if proper reginimal and medicinal precautions be taken, the gouty patient may be saved from such an alternative, and the disease, instead of increasing in intensity, may be gradually mitigated, and probably interfere but little with the comforts of life.

TREATMENT.—The subject of the treatment of Gout naturally divides itself into, first, the treatment of the articular inflammation; secondly, the management of the gouty subject during the intervals of the attacks; and, thirdly, the treatment of the complications and irregular manifestations of the disease. Under the head of Treatment we shall discuss not only the medicines which it may be necessary to administer, but likewise the dietetic and reginimal management.

Treatment of Acute Gout.—Let us first examine if there is any necessity for giving medicine at all, and whether or not it is prudent to leave the joint disease to pursue its own course uninfluenced by any drug.

Cases are now and then met with in which the affection has been left to itself, and several such have come before me. From the opportunities thus afforded, I have ascertained that many of the early and slighter attacks of Gout will subside in a few days, provided the patient is moderately careful in diet; but that, if the usual mode of living is indulged in, the attack may be prolonged, even to many weeks or months; or, if slight remissions take place from time to time, they are soon succeeded by exacerbations, until at last the patient's general health gives way, the appetite fails, and thus, under a necessarily altered diet, the disease exhausts itself: even then, the attack may last a long time, as is likewise the case under homœopathic treatment, which, if honestly practised, and with the use of infinitesimal doses, is, I should imagine, exactly equivalent to the non-exhibition of medicines.

When the articular inflammation is allowed to run its own course, and has been endured for a long time, it leaves a considerable amount of injury in the affected parts, the bloodvessels become weakened, the distension of veins and the local œdema remain, and the joints are left in a condition liable to take on unhealthy action from trifling constitutional disturbances.

Assuming, then, that medicinal treatment can be of real benefit, it is for us to determine the remedial agents best adapted to diminish or cure the inflammation. There is one drug which has an undoubted influence in controlling gouty inflammation, and its action in articular Gout appears as marked as that of cinchona bark in the cure of ague: this remedy is colchicum. It signifies not what part of the colchicum plant is taken, whether the corm, the seeds, or the flowers, for the same principle pervades the whole plant; neither does it signify what preparations are made use of, whether the wine, the tincture, or the extract, provided equivalent doses be administered, for the effects of all are the same.

Colchicum, as before stated, has a direct controlling power over the joint disease, and I cannot call to mind a single instance in which its influence was not well marked, although in many cases a question may arise as to the propriety of its exhibition. Colchicum in full doses produces a marked sedative effect upon the nervous and vascular systems; it has likewise a distinct influence upon the intestinal canal; and if continued too long and in too large doses, causes tormina, and a very troublesome form of diarrhœa. It also produces a peculiar change in the fæcal excretions, so that those accustomed to its use can detect it by this circumstance alone, even when otherwise unaware of the exhibition of the drug: this alteration in the alvine excretions is probably due to the influence of colchicum upon the secreting apparatus of the bowels or their appendages, more especially the liver and pancreas. Colchicum has generally been supposed to cause a more copious flow of urine, and to favour the elimination of its solid constituents; but of this there will be occasion to speak further on.

Although colchicum causes purging, still its peculiar influence is quite apart from this effect. Occasionally an almost magical change is produced by a single large dose, without the appearance of the least increase in the secretion from any organ, the effect being manifested in the rapid subsidence of the pain and other symptoms of the joint inflammation; and simple purging, even though copious, will often fail to produce any notable effect under the same circumstances. I am of opinion that, in articular Gout, colchicum may be advantageously administered during the time that the inflammatory symptoms are present; and the dose of the wine of colchicum may be from ten to twenty or even twenty-five minims repeated every six hours. Colchicum given in the above manner will of itself be sufficient in most cases to cut short the gouty attack, and I have often depended on it alone; but, at the same time, in the majority of cases, it is advantageous to combine it with other remedies, which must necessarily vary in different cases. From what has been stated of the condition of the blood and of the urine, it will at once appear that some moderate alkaline plan of treatment is likely to prove advantageous, both for the purpose of increasing the alkaline state of these fluids, and also to keep in solution the salt of uric acid, which is liable to be deposited in the cartilaginous and ligamentous tissues. There can be no doubt of the value of alkaline remedies in the gouty paroxysm, and, in many cases, such salts, given in a freely diluted form, are sufficient of themselves for its removal, and are peculiarly applicable when there are circumstances rendering the administration of colchicum undesirable. Alkalies may be given either in the free state, or combined with carbonic acid, in the form of the carbonates or bicarbonates, or united with some vegetable acid, as the citric, tartaric, or acetic acids. If the stomach is irritated, and an over-secretion of acid be present, then the free alkalies or their carbonates may be administered; but if, on the other hand, there be no such condition, then the salts with the vegetable acids may be used, which produce an alkaline state of the blood and urine, although they do not act as antacids in the stomach. These alkaline remedies not only tend to keep up and restore the normal reaction of the blood, but likewise augment the excretion of urine, and with it the elimination of those solid matters which are unduly retained in that fluid in gouty states of the system. It is important, likewise, to make a selection of the alkali, and unless there are circumstances which render the use of soda desirable, such as an imperfect action of the liver, or a deficient secretion of bile, this alkali is the least fitted for exhibition, as it has much less power than the other fixed alkalies of dissolving or holding in solution uric acid. The salts of potash are, in the majority of cases, more suitable than the salts of soda, as they not only exert a much greater solvent action upon urate of soda, but likewise augment in a greater degree the excretion of the urine. A third fixed alkali or its salts can be employed in lieu of soda or potash, namely, lithia, an alkali now able to be procured in quantities sufficient for medicinal use.

Besides the administration of alkaline remedies and the cautious use of colchicum, it is important in attacks of acute Gout to attend to the state of the intestinal canal and the skin. If the bowels be confined, some aperient must be given, and the selection of the drug should depend upon the peculiarities of the patient. If mere constipation exists, a simple purgative, as the compound colocynth extract, may be administered at night, followed by a saline aperient, as a seidlitz powder, or the effervescing citro-tartrate of soda and magnesia; or if a more active dose be required, the common black draught can be substituted for these latter. If, however, the portal system shows evidence of congestion, and the function of the liver is disordered, some more powerful cholagogue will be useful, especially if the patient has been in the habit of taking purgatives. A small amount of blue pill or of calomel can be combined with the colocynth, or podophylline, in quarter or half-grain doses, may be substituted for the mercurial. It should be borne in mind that, in gouty habits, mercurials must be used with great caution, as in many cases there is a considerable susceptibility to their action, and very unpleasant consequences may follow their administration in repeated doses.

Saline purgatives are very desirable; many of them act remotely as antacids, and all tend to relieve portal congestion. A very useful combination, and one often employed in acute attacks of Gout, is a draught containing sulphate and carbonate of magnesia, to which colchicum alone, or bicarbonate of potash and colchicum, may be added.

The function of the skin, if very defective, may be promoted by the use of a hot-air or vapour bath, and at the same time the acetate of ammonia may be given, combined with other remedies.

The action of the kidneys is usually sufficiently promoted by the saline treatment, especially if accompanied by the free use of diluents.

Blood-letting, in the form of venesection from the arm, was frequently had recourse to in former times; but this practice is now almost abandoned. The use of small bleedings has still perhaps a few advocates, and of such treatment I had much experience some years since. There can be no doubt that in some cases of very acute Gout, especially when many joints are implicated and fever runs high, speedy and marked relief is procured by taking a small amount of blood from the arm; but it is questionable whether even in such instances it would not ultimately have been better for the patient to have obtained the relief a little more slowly, and without the loss of so valuable a fluid as the blood. The doubtful advantage of the practice will be more apparent when it is stated that everything that produces lowering of the vital powers tends to engraft the disease more permanently upon the system.

A question now arises,—Although general blood-letting is undesirable, should not local depletion be resorted to? The appearance of a joint when acutely inflamed, the state of tension and redness, the high temperature, and the exquisite pain, all seem to point to the necessity of, or at least the advantage likely to accrue from, the abstraction of blood from the part; and the indication has often been acted upon. My own experience quite accords with that of former observers as to the danger of this practice, and several instances have come under my notice of considerable and irremediable injury which has resulted from the use of leeches in these cases. I have frequently seen great-toe joints stiffened after a few attacks, when local depletion has been resorted to, and within the last eighteen months two remarkable cases, in which the patients have completely lost the use of both knee-joints from two or three attacks only: in both instances leeches had been applied very freely; in one, more than thirty to each joint. I can with confidence warn those engaged in the treatment of an acutely-inflamed gouty joint never to have resort to this mode of combating the disease. It would seem that the abstraction of blood from the joint allows or favours the free deposition of urate of soda in the tissues, and thus the ligaments become rigid, and ankylosis ensues. Although more or less stiffness is not infrequently seen as the result of long-continued gouty action in a joint where no local depletion has been employed, still, as a rule, the free movement of a joint is but little impaired by even numerous attacks of acute Gout.

As it has been shown that leeches should not be made use of in the joint affection, the next point is to consider whether any or what local remedies may be advantageously employed. If the pain and redness are slight, all that is necessary is to cover the part with flannel or some other light and warm clothing: this precaution is simply for the purpose of avoiding the chance of a chill. Should, however, the inflammation be very intense and the suffering great, carded cotton should be wrapped around the joints principally affected, and oil silk or gutta-percha sheeting so applied, that the moisture is retained, and by this means a kind of vapour bath is formed. Some care is necessary to ensure the complete closure of the oil silk, so as to prevent the escape of vapour; for, unless this is effected, the warm covering of cotton, instead of giving relief, heats and augments the pain.

As some patients are very intolerant of pain, it is at times desirable to apply anodyne remedies, and the most efficacious are belladonna and opium. I prefer a solution of atropia and morphia, dissolved in spirit and water, in the proportion of one grain of atropia and eight grains of hydrochlorate of morphia to the fluid ounce; a small piece of lint may be dipped in the solution and placed on the part, the oil silk being employed as above described. The tincture of belladonna and of opium may be used, but the solution of the alkaloids is much more cleanly and elegant. Aconite and its alkaloid have been proposed for lulling pain in Gout; but when strong, they may cause irritation of the skin.

Blisters have been used with advantage, when there exists great want of power in the system; possibly their value in chronic and asthenic Gout may be in part due to the fact of the serum withdrawing some of the morbid matter from the affected joint.

We have alluded above to the administration of colchicum, and mentioned some of the symptoms which may arise from its administration in full medicinal doses, and also its marked influence in controlling gouty inflammation.

Some practitioners have attributed the good effects of the remedy to its action on the bowels. That it often purges when given in full doses, and that it may give rise to a peculiar excretion from the bowels, is true; but it is equally a fact that marked and rapid relief frequently occurs from its exhibition when no appreciable influence on the intestinal canal can be detected; and, on the other hand, free purging can be induced in a gouty patient by other means, without the production of relief to the local inflammation. It may hence be safely inferred, that the peculiar influence of colchicum does not result from its purgative action. Others have been disposed to attribute the beneficial influence of colchicum to its action on the kidneys, and have regarded it as a diuretic, which not only causes an increased elimination of the watery portion of the urinary excretion, but likewise of the solid constituents, and more especially the uric acid. If these properties were possessed by colchicum, there would be little difficulty in accounting for its valuable influence in controlling Gout; but, unfortunately, clinical experience does not favour these views. It is true that some observers, as Dr. Christison and Dr. J. McGregor Maclagan and Professor Chelius, have made observations which at first sight would render it probable that colchicum increased the solid excretion, but as only single specimens of urine were taken, and no reference made to the total elimination in the twenty-four hours, a serious source of fallacy existed; the urine after the administration of colchicum might, it is true, have been higher in specific gravity and richer in urates, but this circumstance may have been due to a diminished secretion. Chelius's observations were made on patients recovering from gouty attacks, in whom it is not uncommon to find, for many days, a gradually increasing amount of uric acid without the administration of any medicine. From numerous observations[1] made some years since on the influence of colchicum upon the secretion of urine in gouty and other cases, I arrived at the conclusion that, in health, colchicum diminishes rather than increases the excretion of uric acid and urea by the kidneys, and that the elimination of the watery portion of the urine is often lessened, more especially when purging is caused by the remedy. It is probable that the statements as to the increase of uric acid have arisen from the fact that the analyses have been made on urine passed at some one period of the day only.

Having failed to discover any visible alteration in the principal secretions produced by the administration of colchicum, it is necessary to seek some other mode of explaining its action. That it is a sedative to the vascular system is a well-known fact, which has been fully proved by clinical experience; in subjects with weak hearts it causes temporary intermission of the pulse. Dr. Maclagan found on two occasions twenty minims of the tincture lowered the number of beats from eighty-seven to sixty-five and from eighty-four to sixty-two per minute respectively. This controlling power exerted upon the circulation, although it may explain to some extent the relief experienced from the drug, still is quite unable to elucidate the whole; for if the sedative action were the only effect, colchicum should be equally efficacious in acute rheumatism as in Gout; but that it is not so has been proved beyond doubt. Another explanation of the effects of colchicum has been proposed; namely, that its action is chiefly exerted upon certain tissues of the body, especially the ligamentous and cartilaginous, in the same manner as other remedies are known to affect particular organs, as belladonna the pupil of the eye, digitalis the heart, and so on; but the same objections hold good here as in the former case, for the action of the drug should be equally potent in controlling inflammation of the same tissues when not gouty in its character.

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[1] Medico-Chirurgical Transactions, vol. xli. 1858.

Treatment of Chronic Gout.—The treatment of the acute paroxysm having been sufficiently described, it remains for us to speak of the manner in which the chronic conditions of the disease require to be managed; if in acute Gout it is necessary to make the treatment dependent upon the state of the system and the idiosyncrasy of the patient, it is even still more so when the chronic forms are prescribed for. We have seen that a gouty fit, whether it occurs in the strong and robust or in the weak and spare habit, is dependent on the same proximate cause; yet that it may be excited by various circumstances in different individuals, for in one patient the state of the digestive organs, in a second the function of the skin, and in a third the secretion of the kidneys, may be principally at fault: and all these considerations must be taken into account when called upon to treat any one labouring under chronic Gout.

Value of Colchicum.—Colchicum is found equally efficacious in subduing the exacerbations in chronic Gout as in combating the early fits in the acute disease, due regard being paid to the strength of the patient, and the dose regulated accordingly.

It has been asserted, and the opinion is a very prevalent one, that the use of colchicum in the acute disease tends to cause the attacks to recur more frequently, and to induce a chronic state of the malady; but there are no good grounds for such an idea, unless the remedy has been much abused. It must be remembered that Gout, even when left to run its own course, and quite independent of medicinal treatment, has a powerful tendency to return, and the natural course of the disease should not be confounded with the effects of any treatment which may have been pursued.

It is important to disabuse the minds of both the profession and public of the prejudice against the guarded use of colchicum, as the permanent danger caused by allowing the inflammation to linger for a long period is far greater than any injury which the proper use of colchicum can entail. It is not improbable, if an attack of acute Gout is allowed to run a long course, that, at the termination of the fit, the patient is for a time more free from the disease; *i.e.*, the blood is purer than if the inflammation had been simply arrested without any care having been taken to rid the system of the morbid matter. Although colchicum given alone has a powerful influence in diminishing the subacute inflammations in chronic gouty cases, yet it may often be very advantageously combined with other medicines, and, amongst these, that which claims the first notice is guaiacum.

Value of Guaiacum.—This resin may be given either in the form of the mixture of guaiacum of the Pharmacopœia, in which the powdered resin is kept in a state of suspension by means of the acacia mucilage, or as a powder combined with aromatics, or in many instances, still more advantageously, as the ammoniated tincture of guaiacum made up into a draught.

Guaiacum sometimes acts on the mucous membrane of the alimentary canal as a purgative, but this occurs less frequently with the ammoniated tincture than with the powdered resin. This aperient action is often rather useful than not; but if it is not desirable, it may usually be prevented by the addition of a minim or two of laudanum to each dose. The resin evidently becomes absorbed, at least in part, and after it has entered the circulation acts as a stimulant to the smaller arteries and capillary system of vessels. It often promotes the function of the skin, and clinical experience appears to show that it has a specific effect upon the fibrous and ligamentous tissues, as well as on the mucous surfaces; it also increases the warmth of the extremities, and relieves pain connected with a languid circulation. Guaiacum may be administered for a long period of time without injury; I have had patients under my care who have taken it for a whole year. Within the last few years I have given this drug extensively, and with great advantage; it is especially useful in the asthenic Gout of old subjects, but to young patients it may also be given with benefit.

Value of Iodide of Potassium.—Another remedy of service in chronic Gout is the iodide of potassium. This salt undoubtedly possesses great power in controlling inflammation of fibrous tissues; its action on the periosteum is very marked in the case of nodes, also in painful neuralgic affections dependent upon an inflammatory state of the nerve coverings; it is more especially useful when the pains are increased at night and by the heat of bed. It is also useful in removing the recent thickening of the tissues around joints, but proof is still wanting of its possessing any power of causing the absorption of urate of soda.

In gouty inflammation, when fluid has been thrown into the cavities of the joints, and has been slow of absorption, the administration of the iodide of potassium has often appeared to be attended with great advantage.

Cinchona Bark and Quinine.—The preparations of bark and quinine possess an undoubted power of controlling inflammation, and within the last three or four years I have largely employed them for this purpose. With regard to the action of quinine, there are certain observations in relation to its physiological action which are of interest, and may also prove of therapeutic value.

Dr. Ranke has stated that quinine has the power of diminishing the amount of uric acid in the urine. To prove this, Dr. Ranke gave in one dose twenty grains of sulphate of quinine to a patient, and found that the excretion of the acid was only one-half the average: the influence of the dose continued for about two days.

In my own observations the average of the excreted uric acid during these days was but slightly under that which it had been before the quinine was given. In one instance, for example, the average of uric acid in the urine for two days was 5·89 grains when no quinine was exhibited, and 5·37 grains for three days when the patient was taking eighteen grains of the sulphate each day, in divided doses. Assuming that Dr. Ranke's statement is correct, and that the elimination of uric acid is much lessened for two days after the dose, it is a matter of much interest to inquire if the effect is due to a diminished formation, or defective excretion from the kidneys. From my observations, I was inclined to ascribe the effect to the sudden and powerful impression of the drug upon the nervous system influencing the excretion of uric acid, and not to any decrease in its formation in the system. I shall, however, be unwilling to offer a strong opinion upon the subject at present, as I consider that further experiments are required. Quinine may be beneficial in controlling gouty inflammation, whether it produces one or other effect: of the influence of colchicum there cannot exist a doubt; still it has not been proved either to augment or diminish in any marked degree the elimination or formation of uric acid.

Yellow cinchona bark has been used in lieu of quinine, and where there is great vascular debility, the astringent principle of the bark appears to be of service.

It is advantageous to unite small doses of colchicum to the quinine; and when using the former drug in large doses, the addition of the latter is of service in preventing depression of the nervous system.

Constitutional Treatment of Chronic Gout.—Although due attention to the inflammation of the joints is of great importance in the management of chronic Gout, there is another object to be steadily kept in view, namely, the removal of the morbid condition of the blood, and the solubility of any uric acid which may fail to be eliminated.

It has already been shown that uric acid is thrown out entirely, or almost entirely, by the kidneys, and it has also been demonstrated that in Gout there is always some diminution of the uric-acid eliminating power, and often an almost entire suppression of this function. It follows from this, that one great object must be to endeavour to increase this excreting power, and several remedies may be made use of to effect this, amongst which alkalies and salines stand out prominently.

Value of Alkalies and Salines.—These agents have long enjoyed favour in the treatment of chronic Gout, and not without reason; sometimes they are given in the form of ordinary medicine, sometimes in the form of natural or artificial mineral waters. It may be observed here that as chronic Gout is a disease which has usually been many years in becoming fully established in the system, so it is one in which benefit cannot be expected, except from a long-continued perseverance in some judicious plan of treatment; and when a mere exacerbation of inflammation has subsided, the cure must not be thought to be effected. Under these circumstances it is most desirable that the plan adopted should be simple, and neither disagreeable nor troublesome; and it will be found in practice that a patient will often persevere for an almost indefinite time with the use of mineral waters, when he would refuse treatment by the ordinary mode of administering drugs.

The alkalies and alkaline earths most commonly ordered are the salts of potash, soda, lithia, magnesia, and lime; and these are usually combined with carbonic or some vegetable acid, and occasionally with phosphoric acid.

If these bases are given in the caustic state, they act as direct antacids, and many of them influence the mucous membranes as sedatives, or, in strong doses, as irritants: hence potash, soda, and lithia are seldom given in the free state, unless there are special indications for their employment. When carbonates or bicarbonates of the bases are administered, the antacid effect is equally produced; but if combined with the vegetable acids, as the citric or tartaric, the alkalies lose their power of neutralizing acidity in the stomach. After absorption into the blood, and elimination by the kidneys, the alkaline reaction is equally produced by the vegetable salts as by the carbonates or free alkalies, for the acid is broken up *in transitu*, and the base eliminated in the form of a carbonate. It is, therefore, desirable to select either a carbonate or neutral salt, according as it is thought advisable or not to produce an alkaline effect upon the stomach.

In making a selection of the base, several circumstances must be taken into consideration, and especially the organs and functions peculiarly influenced by the different alkalies and earths.

Potash and its salts act especially on the kidneys, causing not only an alkaline state of the urine, but usually a marked increase in the secretion itself.

Observation has repeatedly shown that potash has a marked effect in augmenting the quantity of urine; its effect on the excretion of the different organic solids has not been clearly made out; no uniform results have been obtained sufficient to show its influence upon the uric acid; some experiments appear to indicate an increased, some a decreased excretion of this acid, and others, again, that it was unaffected. There are many difficulties in the investigation; in alkaline urine, uric acid is soon decomposed, and if the quantity of urine is much increased, a portion of it may be lost in the analysis. Potash and other alkalies may act in more than one way: not only may they increase the elimination of uric acid, but they probably facilitate its destruction in the system, and certainly aid in imparting solubility to it in the blood.

Soda salts have less influence upon the kidneys; they act less powerfully as solvents of uric acid; but, on the other hand, they appear to aid the secretion of bile, or act as hepatic alteratives. They are indicated in gouty cases accompanied with marked derangement of the liver.

Lithia salts have only been used as internal remedies during the last six years. They are active diuretics, more active than salts of potash; they are also powerful solvents of uric acid, and on account of the very small equivalent of the metal, caustic lithia or its carbonate possesses great neutralizing power for acids. The urate or lithate of lithia is by far the most soluble of all the salts of uric acid.

When first introduced by the author as a remedy for Gout, lithia was supposed to exist only in a few minerals, but more recently its presence has been discovered in a variety of substances. It has been found to be a constituent of the human body, of many plants, and can be shown by means of the spectrum analysis in the ashes of the blood, and even of a cigar; it is found, also, in the waters of several mineral springs, especially those of Baden-Baden; also in the springs of Carlsbad, Aix-la-Chapelle, Marienbad, Vichy, &c.

Salts of lithia, especially the carbonate and citrate, have now been employed extensively, and apparently with considerable success. It is, of course, a matter of extreme difficulty to form a very strong opinion upon the value of any drug, especially when the effects are not at once evident to the senses, and are long in being produced; but lithia appears to have many desirable qualities; if the opinion of patients can be relied upon, lithia salts taken for a long period in a very dilute form have the power of preventing gouty paroxysms in chronic cases, and some evidence has been afforded of their power of rendering joints more moveable, and of causing some solution and absorption of chalky matter which has been already deposited. Lithia salts certainly act rapidly and powerfully in preventing deposition of urates and uric acid in the urine, and in the calculous tendencies of many gouty subjects must necessarily prove of much value.

The dose of carbonate of lithia may be from five to ten grains dissolved in aerated water, or the citrate may be given in doses of from eight to twelve grains or more.

Before leaving the subject of the alkalies and their salts, it may be useful to allude to the value of administering these preparations in a very diluted form. Many soluble salts, if given in the form of concentrated solutions, will act as purgatives, whereas if very freely diluted they produce diuresis; and it must be at once evident to any one who considers the subject, that the introduction of a large amount of fluid into the system has the effect of rendering the blood more capable of holding sparingly soluble matters in solution, and of augmenting the various secretions from the body, especially the urine, and hence of facilitating the expulsion of any such matters from the system. It is necessary also to select proper times for the exhibition of these remedies; as the object is to have them rapidly absorbed, they should be given on an empty stomach, at least an hour before food; if taken at or soon after a meal, they are apt to cause distension and discomfort, and at the same time they are very slowly absorbed: any amount of free alkali, when taken at the time that digestion is going on, impedes the process very seriously by neutralizing the free acid of the gastric fluid. This is a fact too frequently overlooked in practice.

Small doses of salines, if their use is long persevered in, are preferable to large ones, as they produce no disturbance of the digestive process, act more freely on the kidneys and skin, and are not likely to cause debility.

Magnesia and its carbonates have been long used in the treatment of chronic Gout, and there can be no doubt that these salts are of value in many cases. Magnesia forms a moderately soluble salt with uric acid; it is also an alkali, and acts both as a direct and remote antacid; it is useful as an adjunct, and especially in instances in which there is great acidity in the intestinal canal, and at the same time a sluggish state of the bowels. The salt formed in the stomach by its union with the acid produces a purgative effect, which tends to relieve the portal circulation, and often aids indirectly the function of the kidneys. Carbonate of magnesia dissolved in excess of carbonic acid is an elegant form of administering the remedy.

The lime salts offer no special advantage. Lime water may be employed as an antacid if there is much tendency to diarrhœa, as the salts of lime have a constipating effect.

There are some other salts, not yet alluded to, whose virtue depends partly on the acid contained in them; for example, the phosphates of soda and ammonia.

Both these salts increase the solubility of the urate of soda, and may be used with advantage in certain instances. The phosphate of ammonia has been employed in many cases of chronic Gout, and clinical observation appears to show that it is useful in preventing paroxysms, probably by keeping the blood in a purer state.

The result of my own experience of the use of the phosphate of ammonia is favourable; it is especially indicated in cases in which the circulation is feeble, and the function of the skin impaired: this remark applies equally to other ammoniacal salts.

Treatment of Errors of the Digestive Function.—As the stomach and other parts of the digestive apparatus are generally affected in chronic forms of Gout, it is of importance that their condition should be carefully attended to; purgatives, stomachics, and alteratives are the remedies resorted to for correcting any morbid state of these organs.

Value of Purgatives.—It has been already stated that free purgation alone will not rapidly cure gouty inflammation, and that it is impossible to explain the effects of colchicum upon this idea; that, for example, sulphate of magnesia, although it causes a free watery action, will not relieve in the same manner as colchicum, even when the latter drug produces no appreciable action upon the bowels. Purgatives, however, are often of much value, especially when there is portal congestion present, and the occasional use of the compound colocynth extract, or some such preparation, is usually of great advantage. Mercurials should, as a rule, be avoided, as they are peculiarly prone to cause ptyalism in gouty subjects, and as their frequent use lowers the powers of the system.

Value of Stomachics and Tonics.—If there exists an irritative form of dyspepsia, accompanied with flatulence, acidity, and heartburn, this is generally relieved by the administration of free or carbonated alkalies; if pain or palpitation is present, a few drops of hydrocyanic acid may be added to each dose, taking care to relieve any sluggish condition of the liver and bowels. These alkalies are often advantageously combined with some bitter stomachic, as the juice or extract of taraxacum; or, if there appears to be great want of tone, the more powerful bitters, as chamomile, gentian, chiretta, quassia, &c. &c. If much flatulence be present, then ginger or capsicum may be likewise added.

Ash-leaves in the form of an infusion have also been recommended, and clinical proof afforded of their efficiency in chronic Gout. I have frequently made use of them, and with advantage; but the amount of infusion taken each day has always been considerable, and the action of the diluent must not be forgotten.

Value of Ferruginous Preparations.—In the majority of instances iron salts are not indicated, but at times cases are met with in which their administration is attended with striking benefit. They prove most useful when the blood is impoverished from the diminution of the red corpuscles, and the circulation enfeebled from weakness of the walls of the heart, and where the whole nervous system has become exhausted. It is often a good plan to combine these with small doses of the extract of colchicum.

The selection of the ferruginous preparation should be made according to the peculiar requirements of the patient: if only the hæmatinic property is wanted, reduced iron (ferrum reductum) may be prescribed; if the astringent influence is required, the sulphate may be made use of.

Value of Diaphoretics or Sudorifics.—Friction of the skin must not be overlooked in the treatment of chronic gouty cases; we must remember, that although there is no elimination of uric acid from the healthy surface, still the skin gives off a large amount of some other acid, which, when retained, renders the blood serum less alkaline.

The salts of ammonia are useful when the skin is particularly in fault, especially those in which the alkali is combined with a vegetable acid, such as acetic acid; but it is better in most cases to promote the due performance of the cutaneous function by insisting upon a sufficiency of exercise, and the free use of water to the skin, combined with friction, and wearing flannel next to the skin.

Hot-air and vapour baths are also valuable, and, when it can be borne, the Turkish bath occasionally; hot salt-water baths may also be made use of.

Treatment of the Local Affection.—One of the slighter forms of inconvenience, arising from long-continued gouty inflammation in any part, is the production of œdema, a symptom evidently depending on local debility or weakness of the vessels of the affected parts, usually the lower extremities: this is often much increased by disease of the kidneys, or sometimes of the heart. When the swelling is dependent simply on local weakness, it is best treated by the use of some mechanical support, as the elastic stocking and slight friction, with or without some stimulating and lubricating application; the limb should be elevated, so as to facilitate the free return of blood to the heart. Œdema depending on a kidney or cardiac disease must be specially treated.

A far more distressing complication, and one far more difficult to deal with, is that arising from chalk-stones, and the distortion and rigidity of the joints produced by deposition within the structure of the articulations.

If chalk-stones appear on superficial parts, they may be of little inconvenience, and are often spontaneously removed: for example, if the nodules on the ears of gouty patients are observed for some few years, a considerable change is generally noticed; they may escape from rupture of the cutis, or if they increase in size, so as to cause any inconvenience, they may be punctured, and by this means dispersed, the contents escaping either in the semi-liquid or solid state.

There is no danger attending an operation upon the ears, nor in many other situations, when the chalky concretions are small, and especially when in a liquid state and very superficial, provided the patient is at the time in a tolerable condition of health; but the case is far otherwise if the chalk-stones are large and solidified, and especially if their ramifications are deep-seated. In such cases the removal is apt to be attended with serious and even fatal consequences, arising from the extreme difficulty in the healing of the sore, or the supervention of a low form of erysipelas. The greater the amount of kidney affection, the greater the danger in operating. Not infrequently, when the deposits approach the surface, they burst spontaneously, and abscesses are then formed: these are often very difficult to heal on account of the matter being deep-seated. I have known cases in which such abscesses acted as a kind of safety-valve, and their closure has been immediately followed by a paroxysm of gouty inflammation. The best mode of treating these abscesses is to keep upon them a water dressing, as long as there is a free flow of the matter; then to have them dressed with some stimulating ointment, and occasionally either to apply a lotion of sulphate of zinc, or touch the surface with a stick of lunar caustic. Now and then, if the opening becomes nearly closed, at the time that there is a large accumulation of the urate of soda behind, it is advisable to enlarge the opening by a slight incision.

It has been thought by some physicians, both in ancient and modern times, that the deposits of urate of soda which occur in Gout are capable of being dissipated by external applications, and alkaline preparations have usually been employed for this purpose, such as solutions of potash, or soda, or lime. I have in some cases had compresses, steeped in a solution of carbonate of lithia, kept on the parts for a long time, and patients have assured me that they have found decided benefit from them; but although such a solution exerts a very powerful solvent action upon the deposits when removed from the body, it is difficult to imagine how they can act through the skin: but as such treatment can do no harm, it is worthy of a further trial.

When joints are much stiffened from gouty inflammation, considerable relief is obtained from the use of a blistering liquid. This appears more useful, and, upon the whole, less annoying to the patient than iodine paint; and it usually removes any effused liquid, and chronic inflammatory action, which may be lingering about the part. After all tenderness has disappeared, movement may be attempted, very gently at first, but gradually increased; the joints may also be rubbed with some slightly stimulating liniment, or with strong salt and water. In the treatment of these cases it must always be borne in mind, that the rigidity is often due to interstitial deposit in the ligaments, and that a restoration of these structures to a healthy state can hardly be hoped for: still, as this condition may often be complicated with a chronic inflammatory action, it is always advisable to attempt the removal of such complications.

Treatment of the Irregular Forms of Gout.—The treatment of irregular Gout must necessarily be very different in different cases, and it is a matter of extreme difficulty to lay down any precise regulations for the guidance of the practitioner: however, a few general rules may be advantageously given.

Should metastasis take place to any important organ, it seriously implicates its functions, and therefore our main object must be to take such steps as will restore this function; and, as the metastasis usually follows the sudden suppression of gouty inflammation of some joint, the one very essential part of the treatment consists in the endeavour to bring back articular inflammation. This object is best effected by the application of heat or counter-irritation to the extremities, as by hot bottles, sinapisms, &c. At times we may treat the part itself, especially if there is evidence of inflammatory action existing in it: this may be effected by leeches, blisters, and mustard poultices; local depletion, however, is rarely necessary. The peculiar condition of the system, or the existence of the gouty diathesis, must not be overlooked. A question of no little importance at once arises when the diathetic condition is considered; it is that which relates to the value of colchicum in irregular Gout. Sir Henry Holland is of opinion that it can be employed with advantage, and as far as my own experience goes I quite coincide with him, although unable to explain its action. This want of knowledge applies with equal force to the action of colchicum in the genuine articular form of the disease.

In metastatic Gout of the heart and lungs, cardiac stimulants, as ammonia, ether, and other anti-spasmodics, are especially indicated. If the bladder is affected, belladonna and henbane may be advantageously used to diminish spasm; in short, remedies directly influencing the implicated organ may in all cases be resorted to, at the same time that the means tending to reinduce the articular inflammation should not be neglected.

Value of Mineral Waters in Gout.—Mineral springs are frequently resorted to by gouty subjects, and it is important that the medical man should know when to advise and when to oppose such a step, and likewise the waters most suitable for different cases.

Although it cannot be denied that many patients receive great benefit from the proper administration and use of these waters, still it must be allowed that their action is not always beneficial, and that in some cases it is very injurious.

All mineral waters have one action in common; it is that of water itself; and there is little doubt that the value of this agent when properly employed is considerable. The waters of some of the mineral springs of great reputation contain little foreign matter, and must owe most of their efficacy to the water alone. The other waters employed in gouty cases contain either alkaline carbonates, chlorides, or sulphates. Some of the waters are impregnated with sulphuretted hydrogen, and another class owe the chief of their powers to the iron which enters into their composition. Many of the springs are of an elevated temperature; some of mean heat; others cold.

All mineral waters rich in saline matters, if taken too freely, usually set up a febrile disturbance or crisis; the system becomes oppressed, there is a feeling of heaviness, languor, or agitation, and this is followed by loss of appetite, thirst, a furred tongue, and heat of skin, sometimes by vomiting and diarrhœa. Such symptoms are probably due to the blood becoming saturated with the saline matters, from the excreting organs being unequal to the task of eliminating the whole quantity introduced during the treatment.

The different springs of Vichy are all rich in carbonate or bicarbonate of soda, containing about forty grains to the pint: some have the temperature of 101° Fahr.; others are cold. When taken internally in even moderate doses, they cause the urine to be neutral or alkaline, without affecting the transparency of the fluid; when employed in the form of the bath, the effects appear to be very similar. From the soda contained in them they probably act upon the liver; and from the amount of liquid absorbed, and the temperature at which the water from many of the springs is drunk, they also influence the function of the skin.

Vichy waters appear to be adapted for the treatment of Gout when it occurs in strong subjects in whom the function of the liver and digestive organs is at fault, and are contra-indicated in very chronic cases, especially if there is a tendency to the rapid formation of chalk-stones, or if the powers of the system have become much enfeebled: my own experience is that, in this latter class of cases, they rather tend to favour the formation of these concretions.

The internal exhibition of the waters is usually accompanied with the use of the bath; if they disturb the stomach, the bath alone should be employed.

Wiesbaden waters contain a large amount of chloride of sodium, and are of a high temperature, 160° Fahr. They are less debilitating than Vichy waters, and more stimulating to the various functions: they consequently are more adapted for cases in which the circulation is sluggish and the secretions deficient; also in cases of rigidity from thickening of the textures. They are powerless in removing any solid deposition of urate of soda.

Aix-la-Chapelle is frequently resorted to; the waters are slightly saline compared with those of Wiesbaden, high in temperature, 135° Fahr., and, in addition to chloride of sodium, contain some carbonate and sulphide of sodium, with free sulphuretted hydrogen. They act as stimulants to the secreting organs, and more especially to the skin; they are indicated in cases in which the skin is in fault, and they have also been found useful in removing rigidity of the joints.

The waters of *Aix-à-Savoy* resemble closely, as far as the sulphur is concerned, those of Aix-la-Chapelle.

Carlsbad waters are in great vogue in the treatment of Gout. They are rich in sulphate of soda, and contain likewise carbonate of soda and chloride of sodium; of a high temperature, 167° Fahr. They often cause purgative action, and likewise give activity to the kidneys and skin, and are useful in cases accompanied by deficient action of the bowels, with a congestive state of the liver; but should be avoided by weakly patients.

The waters of *Baden-Baden* are saline, and from recent analysis are said to be rich in lithia. Dr. Ruef has affirmed that they have proved very useful in Gout, and possess the power even of removing visible deposits of urate of soda.

The waters of several other springs, as of *Kissingen*, *Marienbad*, *Homburg*, *Ems*, and several other localities, contain saline matters, and have occasionally been used in the treatment of gouty conditions of the habit.

The waters which possess but little solid matter, and which have acquired a reputation in gouty cases, are those of *Wildbad, Teplitz, Gastein, Buxton,* and *Bath.* They are all of somewhat elevated temperature, are chiefly used in the form of the bath, and appear to be peculiarly adapted for the treatment of the disease in the old and infirm. According to numerous observers, great benefit has been often experienced from their employment.

The following rules may serve as a guide in prescribing the use of mineral waters in gouty cases:—

1. They should not be employed when there exists any appreciable amount of organic disease either of the heart or kidneys.

2. They should be avoided when an acute attack is either present or threatening.

3. The particular water should be selected according to the nature of the case. When the patient is robust, and of full habit, the alkaline springs; when torpidity of the bowels predominates, the purgative waters; when there is a want of vascular action, the saline waters; when the skin is inactive, the sulphur waters; lastly, when debility prevails, then the more simple thermal waters should be chosen.

4. In all cases the use of the waters should be cautiously commenced, and care should be taken not to oppress the stomach by giving too much liquid, nor to induce debility or other injurious effects by allowing too long a sojourn in the bath.

5. In every instance, when practicable, it is advisable to avoid producing the so-called "crisis," for when febrile disturbance is set up in the system, the secretions are checked, and an acute paroxysm of Gout is almost always induced; it is far better to take a prolonged than a too severe course of a mineral water.

Diet and Regimen in Gout.—The diet in the treatment of the different forms of Gout is of great importance, far more so than in the majority of diseases. When the affection is acute in character, and the patient robust, he should be confined for a few days to a diet consisting of little more than farinaceous food and diluents; and this kind of food may be persevered in until the inflammation shows a decided tendency to abate, the thirst diminishes, and the appetite begins to return. Under the term farinaceous food are included bread, arrowroot, sago, tapioca, and such-like substances; to these may be added milk; while water, and toast-and-water, may be indulged in without restraint, provided the liquids are taken upon an empty stomach. Stimulants are scarcely required under these circumstances; but if the patient has been accustomed to live freely, a little brandy may be taken with the solid food; even a moderate indulgence in wine or malt liquor will keep up the gouty inflammation for an almost indefinite period.

When febrile disturbance has abated, a more generous diet may be allowed—at first fish, then fowl or game, and at last ordinary meat. In strong persons it is desirable to keep a moderate curb upon the appetite, for fear of inducing a recurrence of the inflammation.

As soon as possible exercise may be resumed, and it is most desirable that this should be persevered with daily; but if the lower extremities are much affected, there is a fear lest too great an amount at any one time may excite local irritation in parts which have recently been inflamed.

As yet it has been assumed that the gouty attack has occurred in a strong individual, and is of an acute and sthenic kind; but this may not always be the case, for even a first fit may find a patient broken down in constitution, and quite unable to bear the least withdrawal of nourishment or stimulus. Under these circumstances, care must be taken to give such nourishment as the digestive organs can easily assimilate, as beef-tea, strong but plain soups, eggs, milk, &c.; and, when practicable, to confine the stimulus to some distilled spirit, as brandy or whisky, giving these only to the extent of keeping up the action of the heart and the efficiency of the circulation.

A few general rules may be advantageously given, which will serve as a guide for the treatment of gouty patients in general, and apply more especially to such as suffer from the chronic forms of the disease.

It is desirable to regulate the amount of food, so that the system shall be fully nourished, and the strength kept up as much as possible, but anything taken beyond this is decidedly injurious, as it tends to oppress the digestive organs, and induce debility rather than vigour of frame.

As to the character of the solid diet, it may be stated, that every article which causes unpleasant symptoms, recognisable by the patient himself, should be studiously avoided; and hence the less what are termed "made dishes" are partaken of the better: the same remark applies to all rich and highly-spiced food, and to anything that tempts the person to take more than he otherwise would. Articles of animal food of which the texture has been hardened, as salted meats, hams, and so on, are less easy of digestion, and should be discarded, as also veal and pork, which are much less easy of digestion than mutton and good beef; white fish is generally digestible, as also fowl and game.

There should be a due admixture of animal and vegetable food; it is an error to suppose that an animal diet necessarily tends more to the formation of uric acid than a vegetable one. The tortoise, feeding on a simple lettuce, excretes a large quantity of urate of ammonia, far more, in proportion to the weight of the animal, than is excreted by the dog exclusively nourished with meat.

Vegetables, as potatoes, greens, and the like, may be partaken of with advantage; the soluble salts which they contain are of value in keeping up the activity of the secreting organs.

The same remarks hold good with regard to soft fruits when partaken of in moderation, as strawberries, grapes, and oranges; also other fruits when stewed or baked, as apples and pears; but these latter, as likewise plums, and stone fruit in general, should be avoided in a raw state. Extreme moderation should be exercised when saccharine fruits are eaten, as sugar is liable in many subjects to lead to the production of acidity, and hence favour the development of Gout. The same precaution is necessary in reference to the addition of sugar to other articles of diet.

As to beverages, both tea and coffee may be taken if they do not disturb the nervous system. At one time it was supposed that the latter was prophylactic against Gout, seeing that the Turks enjoyed an immunity; but, if strict Mahometans, they do not take alcohol in any shape, and hence avoid its most powerful cause.

It is an important question to decide whether alcohol is to be ever allowed, and, if so, to determine the form which is best adapted for the patient.

All malt liquors should be eschewed, as they almost always cause an increase of dyspepsia, and, if at all strong, have undoubtedly a very powerful influence in inducing the disease and in keeping up a paroxysm.

Strong wines will also prolong an attack to an almost indefinite length of time, and if they are moderately indulged in will often lay the foundation of the gouty diathesis.

The wines to be carefully avoided are port, sherry, madeira, and any in which the fermentation has been checked by the addition of alcohol. If wine is taken at all, that which is best adapted for the majority of patients is a sound claret—one free from sugar and without acidity. When red wine does not agree with the stomach, then hock or moselle may be substituted, or even a light and dry sauterne or chablis.

The beverage best suited for those of a strongly-marked gouty diathesis is undoubtedly French brandy, taken in very limited quantities, and freely diluted with water.

Whisky, hollands, or gin, may in many cases be substituted for brandy; but the two latter should be avoided if there is any appreciable amount of kidney disease, or at least should not be taken without advice. The distilled spirits should only be used at the meal, and from one to three ounces may be daily allowed, the amount depending upon the former habits of the individual.

If Gout has become developed at a very early age, and the youth strongly inherits it, a question arises whether it would not be desirable to advise an entire abstinence from alcoholic drinks. Such a step would be the most likely to check the future progress of the malady.

Exercise must be enjoined, for it is of the highest importance, and without it all our endeavours may prove futile. The kind of exercise must be adapted to the peculiarities of the patient; walking and horse exercise are equally useful, and may be conjoined with advantage.

Fresh air is of great importance, and in many instances a complete change during the winter and spring to some warm and dry climate will enable the patient to escape an attack.

All violent exercise likely to cause exhaustion, all severe mental application and late hours, should be studiously eschewed.

In concluding the subject of the management of Gout, the author's opinions may be thus summed up:—

1. Gout in its acute form is quite as controllable, and as much under the influence of remedies, as any other inflammatory affection. The duration of the paroxysm and the amount of injury to the joints depend much upon the treatment.

2. The more chronic forms of Gout, which are met with in every degree of severity, are likewise under the control of the physician, if not for their radical cure, yet for so much relief as will enable the patient to enjoy life, and prevent further increase of the mischief, so liable to ensue if the disorder is allowed to run its own course, and more especially if recklessly tampered with.

3. As Gout is a disease which is not only apt to return with increased severity, but to acquire a firmer hold on the constitution at each visitation, it is a matter of serious moment to consider whether it may not be prudent in the intervals of the attacks, not only to regulate the diet and regimen, but even to have recourse to means, scarcely to be called medicinal, by which the blood may be kept free from the impurities which lead to the production of the paroxysms.

4. The treatment of Gout founded on Cullen's aphorism, of trusting to patience and flannel, is to be highly deprecated. It may indeed be argued that it is the natural treatment, and that nature is a sure guide; but it must be remembered that man living in a civilized state is not in a normal condition, or in all probability he would never have acquired the disease, and that when suffering from a disorder so acquired, he must be content to have recourse to artificial remedies. If he could entirely lay aside his usual habits, and follow in all respects the dictates of nature, there would probably be little need to seek relief from medicine.

5. Although a plan can be sketched out which is applicable to the majority of cases of Gout, still each individual case not only exhibits its own peculiarities, and becomes a separate study, but likewise demands, in certain respects, a separate treatment. The neglect of this consideration is apt to lead to a mere routine practice, closely bordering on empiricism.

RHEUMATOID ARTHRITIS.

By Alfred Baring Garrod, M.D. F.R.S.

Definition.—A form of inflammation of the joints, accompanied with but little febrile disturbance, and distinguished from gout and rheumatism by its progressive character, by the peculiar morbid changes which it induces, and by the absence of any known morbid state of the blood.

Synonyms.—Rheumatic Gout is the name commonly given to this disease, but equally applied to other joint affections. Dr. Adams uses the term Chronic Rheumatic Arthritis. Dr. Todd included it under Chronic Rheumatism of the Joints. It has been called Nodosity of the Joints by Haygarth and Heberden; Usure des Cartilages articulaires by Cruveilhier; Rheumatism Noueux, by Trousseau and other French writers. The term Rheumatoid Arthritis was applied to this disease by the author in 1858.

History of Rheumatoid Arthritis.—It will be essential to the clear understanding of this subject that some explanation be first given of the name proposed to be employed to designate this disease, and the reason of its adoption; and to give reasons for the rejection of the many terms which have been used at different times. The term "rheumatic gout" is one which is very commonly employed, both by the profession and the public; but it is difficult to arrive at its true significance, seeing that but few have described it as a separate disease: the term has also been used to signify very different diseases. It is not uncommon to hear gouty patients say they are suffering from rheumatic gout, simply because the disease, which for many years was manifested in the feet only, now implicates other joints, as the elbows and hands; in fact, they regard their malady as gout when it is confined to the feet, but as rheumatic gout when it affects the upper extremities. Sometimes the sub-acute forms of true rheumatism are designated as rheumatic gout, and more especially if the smaller joints are the seats of the attack. There exists, however, a third disease, distinct both from gout and rheumatism, to which the name is more frequently applied: it is this malady which we have called Rheumatoid Arthritis; and it this which will engage our attention in the present article.

The name "chronic rheumatic arthritis" has not been employed, partly from the fact that the disease sometimes assumes an acute character,—if this were the only objection, it might be argued that we could designate this form by the name of "acute rheumatic arthritis;" and partly because the name rheumatic arthritis implies that the disease partakes of the nature of true rheumatism, which we believe is not correct. The same objections, of course, apply to the words "chronic rheumatism of the joints."

"Usure des cartilages articulaires" (wasting of the articular cartilages) is an expression limited in its meaning; it only expresses one of the morbid changes which result from the disease.

"Nodosity of the joints," and "rheumatism noueux," express the presence of a frequent alteration in appearance, caused by the affection—one not constantly found.

"Morbus coxæ senilis" could not be used except when one particular joint is implicated, and it is a matter of some doubt whether this form of the disease has the same pathology.

The term "Rheumatoid Arthritis" has been employed for the following reasons:—

The disease is one chiefly affecting the joints, and is of an inflammatory character; hence the name arthritis. It is also one which, at least in its early stages, produces external changes closely resembling those caused by sub-acute forms of rheumatism; but as it can be shown that the nature of the affection is not the same as that of rheumatism, the prefix "rheumatoid" instead of "rheumatic" is sufficiently expressive.

As the word typhoid is allowed for the purpose of designating a form of fever somewhat resembling, but not identical with, typhus, so no objection can be raised to the use of the prefix rheumatoid, when it is intended to signfy that the articular inflammation, although not of the same nature as rheumatism, yet resembles it in some of its characters, and more especially in those which are readily appreciated by the senses.

With regard to the history of our knowledge of Rheumatoid Arthritis little can be said. It is only since the time of Haygarth that it has been looked upon by any pathologist as an independent disease, and even at the present time it is often described under the name of chronic rheumatism, rheumatic gout, &c., and classed as a variety of some other affection.

DESCRIPTION OF RHEUMATOID ARTHRITIS.—Rheumatoid Arthritis is met with either as an acute or as a chronic disease: the former is much less frequent than the latter; in fact, it has only been described within the last few years; it will, therefore, under the circumstances, be desirable to reverse the ordinary mode of treating such subjects, and to describe first the more common form, namely, chronic Rheumatoid Arthritis.

Chronic Rheumatoid Arthritis.—The affection may occur in both sexes, and at almost every age, and its invasion often assumes a form very similar to that about to be described. A young woman has become, from some cause, decidedly out of health; perhaps from menorrhagia or leucorrhœa, or from hæmorrhage during parturition, or from prolonged anxiety or physical fatigue; she is exposed to cold, and, after a few days, feels some pain in the knee; there is slight swelling and tenderness; perhaps the temperature is a little elevated; simple rest to the joint may be followed by relief, or even a cure, for the time, the swelling abating, and the tenderness and pain vanishing; or perhaps this result may have been expedited by the application of a blister, or some other form of counter-irritation.

After a few weeks or months, as the case may be, perhaps from a second chill, another joint, or even that which was previously implicated, becomes affected, and a similar train of symptoms arises, but with this important exception, that, in all probability, the inflammation does not again subside, but continues fixed to the joint, and gradually extends to others. During this time there may be no appreciable constitutional disturbance beyond the general ill-health above noticed, but in some cases dyspepsia or nervous symptoms are exhibited.

The disease, if unchecked, travels over the whole body, affecting almost every articulation of the limbs, and causing much deformity and distortion, from the enlargements and contractions which it produces; nor are its ravages confined to the limbs alone, but other joints may be attacked, especially the temporo-maxillary articulation, causing the closing of the jaw; the upper cervical vertebræ may likewise be involved, and the neck become fixed; and thus at last the patient is rendered crippled and altogether helpless throughout the remainder of life.

The above example is, indeed, one in which the disease has effected all the mischief it is capable of: fortunately, it does not always proceed to this length, but is arrested at some stage or other of its progress, and then only a limited amount of distortion is induced.

The deformities produced by Rheumatoid Arthritis are not altogether characteristic of this disease, for they are produced likewise by chronic gout; but in their advanced conditions the separation of the one disease from the other is usually a matter of no difficulty.

The changes in the arms and hands, when severely affected, are of the following kind:—The elbow is flexed, perhaps at an angle of 35° from full extension; the forearm in a semi-pronated position; the joint is also much enlarged and misshaped, more or less rounded from the alteration and hypertrophy of the heads of the bones, as well as of the soft tissues. The wrists are rigid, almost straight, and scarcely admit of motion in any direction.

The hands are usually thin, from the absorption of fat, and from the wasting of the other soft tissues; the extremities of the phalanges are nodular, as also the heads of the metacarpal bones. The fingers are usually turned outwards, and their joints rigid, often completely fixed.

As a rule, the metacarpo-phalangeal articulations of the fingers are flexed, and the first phalangeal extended, causing the second phalanx to be thrown backwards: the second phalangeal joint is also flexed. One, or even every finger in a hand, may be thus altered. The phalangeal joint of the thumb is usually extended, or bent backwards. Sometimes the nodose condition is well marked, but not infrequently it is but slightly developed; complete dislocation of some joints is occasionally met with. The knee is generally much enlarged and rounded in the same manner and from the same causes as the elbow: it is commonly half flexed. Sometimes there is evidence of liquid effusion, but in the later stages this may be wanting.

When the hip is affected, the thigh becomes flexed, sometimes abducted, with the foot everted; at other times adducted, with the foot turned inwards; not infrequently there is felt over the large joints a sensation as though the bones were loose.

As a rule, the hands become crippled and distorted at an earlier period than the feet. Though frequently attacked, the jaw and neck seldom become fixed.

An affection which, if not identical with Rheumatoid Arthritis, at least closely resembles it, was first made known to the profession by Heberden, under the title of "digitorum nodi," and described by him as consisting of little hard knobs, about the size of a pea, situated upon the ends of the fingers, where they remain through life, being usually attended with little or no pain, and, though they cause but slight inconvenience, are decidedly unsightly. Heberden thought that they had no connexion with gout, seeing that they occur in persons never afflicted by that disease; but Dr. Begbie considers them of a gouty character, as they are frequently met with in that diathesis.

The disease is sometimes confined to the extreme ends of the fingers, but now and then extends to the other small joints. When examined, the nodular feeling and the peculiar appearances are found to be owing to hypertrophy and other alterations in the epiphyses of the phalanges, and, except in position, and the small size of the affected joints, do not appear to differ from the enlargements and distortions above described as occurring in other situations.

Ladies are often much concerned at finding these nodules on their fingers, and are willing to take any amount of trouble to arrest the progress of their formation and to attempt their removal.

Acute Rheumatoid Arthritis.—Now and then cases are met with which, in most of their symptoms, closely resemble acute rheumatism; several joints are attacked, the swelling is considerable, there is distinct increase of temperature of the affected parts, with pain, tenderness, and redness. In these instances, constitutional symptoms, as thirst, loss of appetite, heat of surface, a rapid pulse, and other evidences of febrile excitement, are often observed. There are, however, wanting some of the characteristics of rheumatic fever—namely, the profuse sweating and the proneness to acute inflammation of the internal and external membranes of the heart, so common in acute rheumatism, and likewise the erratic disposition or tendency of the inflammation to fly from joint to joint. Between cases of genuine acute Rheumatoid Arthritis and those of the very chronic varieties there is every intermediate shade of difference.

The only real difficulty in these cases is to determine whether the acute disease is true Rheumatoid Arthritis, or whether it is genuine rheumatism which has acted as the exciting cause of the former affection.

As the acute disease is so little known and recognised by the profession, it may be well to give an illustration, and the following case may be taken as a typical example.

A lady, forty-two years of age, when living in Australia, in the bush, was confined, and being unable to procure a good supply of cow's milk, was induced to nurse her child for a period of twenty months: at the same time she herself had but a very deficient amount of meat. By these means she was reduced to an extremely weak state. After a short time she noticed that some of her joints became affected; at first the knees, then the ankles, afterwards the elbows and wrists, and, lastly, many of the small articulations of the hands. These parts were painful, somewhat swollen, hot, and tender, but the local symptoms were never intense, nor was the constitutional disturbance very great; that is, there was no high degree of febrile excitement. After a few weeks some of the joints were much injured; the knees, although reduced in size from the absorption of the fluid, could neither be fully extended nor flexed, and the patient was soon unable to stand by reason of their rigid condition; the movement of several of the other joints was also limited, although in a less degree. The causes of the debility being removed, the patient soon gained strength and flesh, and the tendency to the joint affection passed off, but not without having inflicted irremoveable injury.

Diseases caused by the Rheumatoid Arthritic Diathesis.—In both gout and rheumatism, symptoms which may be termed irregular manifestations are occasionally met with, and the same holds good in Rheumatoid Arthritis; in other words, structures other than those of the joints, but of a similar nature, may take on the same kind of diseased action. Sometimes these symptoms occur simultaneously with the joint affection; sometimes they alternate with it, or appear to be altogether independent of it. In well-marked cases of this irregular form of the disease, the inflammation has attacked the eyes, ears, or structures of the larynx; producing in the first organ sclerotitis, in the second inflammation of the internal ear, and in the last hoarseness, and a peculiar dry cough, not attributable to pulmonary disease.

Analysis of the Animal Fluids in Rheumatoid Arthritis.—Very little information has been obtained from an examination of the blood, urine, or sweat of patients, suffering from Rheumatoid Arthritis, which is calculated to throw light upon the nature of the disease.

The only analyses of the blood that the writer is cognisant of have been made by himself, and with simply a negative result. If any amount of active inflammation is present, the fibrin is increased, and the clot becomes firm, cupped, and buffed.

The serum has the ordinary properties of the serum of healthy blood, and yields no uric acid.

The analysis of the urine has likewise given negative results.

The perspiration has not been examined, but there is no reason to suppose that any peculiar alteration would be found in it.

MORBID ANATOMY OF RHEUMATOID ARTHRITIS.—The morbid anatomy of this disease has been very elaborately worked out by Dr. Adams, to whose volume the reader is referred for full details of the various changes which take place. In the present article a summary only of the results found in different cases, and different stages of the malady, will be given.

If a joint is examined in an early stage of the disease, when swelling is prominent, a considerable increase of synovial fluid is found, and the joint exhibits the same appearances as in cases of ordinary inflammation; the lining membrane is often red, from over-injection of the bloodvessels. It is not an easy matter to obtain an opportunity of examining joints in this condition, as Rheumatoid Arthritis is seldom fatal, except in its very advanced stages; but the supervention of other maladies sometimes enables us to do so. No deposits of urate of soda are found in any stage of this disease, and I am persuaded that the statements to the contrary are erroneous, and, as yet, there has been certainly no proof given in support of such assertions. Of course it is not impossible that a patient may have had gout in a joint, and that afterwards the same articulation may become the seat of Rheumatoid Arthritis; but this, if indeed it ever occurs, is most rare, and would not in any way favour the idea that deposition of urate of soda is a phenomenon of this form of inflammation.

When the effusion in a joint has been absorbed, the capsular membrane is usually found thickened. In the hip, or shoulder, the round ligament or tendon of the biceps is probably destroyed; inter-articular cartilages are sometimes absorbed; and a case has recently come under my observation in which the articulation of the jaw exhibited this alteration in a very complete degree. If the fluid becomes absorbed before much serious change has taken place in the internal structures of the joint, the ligaments have generally undergone so much lengthening as to allow of unnatural mobility, thus rendering dislocation easy.

From almost the very commencement of the inflammatory action, the articular cartilage begins to suffer, a slow process of absorption takes place, the cartilage appears to split up into fibres, vertical to the surface of the bone; little depressions are observed, and these at length coalesce, and the bone is left in part uncovered; as the disease proceeds, the whole surface may be thus denuded, and as the osseous surfaces are brought into contact with each other in the movements of the articulations, they become polished in a remarkable degree by the friction, and an ivory-like condition, termed eburnation, is produced. Sometimes this eburnation occurs in streaks or patches in the direction of the motion of the joint; sometimes the whole surface may become thus altered. More rarely the cancellated structure of the heads of the bones is exposed by the absorption of the denser matter at their extremities, and the ends of the bones become enlarged and misshapen by the deposition of osseous matter. If the bone is sawn through, it is often found unusually spongy, and contains a large amount of oily matter, from the occurrence of a species of fatty degeneration.

Within the joints vegetations and bands are frequently seen, also foreign bodies of various sizes, some cartilaginous in structure, others having the consistence and texture of bone; and these are usually adherent to the internal surface by ligamentous bands.

CAUSES OF RHEUMATOID ARTHRITIS.—The predisposing causes of Rheumatoid Arthritis may be thus classified:—First, those which are inherent in the patient; secondly, those which arise independently of the individual.

1. *Influences dependent on the Individual.—Hereditary Disposition* does not appear to exert any very special influence, except that children of weakly parents probably inherit their debility. In looking over the histories of a large number of cases, the writer cannot find much evidence of the direct influence of hereditary predisposition; if it exists, it is very much less powerful than in the case of gout. It is not uncommon to find one member of a large family suffering severely from the disease, and the rest entirely free from it.

Sex.—It is commonly thought that women are more liable to Rheumatoid Arthritis than men. M. Trousseau speaks of the affection as very rare among men, very frequent amongst women. Women are doubtless very prone to be attacked, as they are more likely to be subjected to the predisposing causes, especially irregularities of the uterine function; and it would appear that deranged menstruation, independent of hæmorrhage, predisposes to the disease. Men, however, are by no means free, and some of the most severe cases are found among them.

Age.—Rheumatoid Arthritis may occur at almost any age. I have seen it in its worst form in children of ten and twelve years of age, and I have also seen it commence in very old people above seventy years.

Individuals of weak frame, whose circulation is languid, and whose extremities are habitually cold, are more liable to the disease than others; and it should be mentioned that patients having a tubercular diathesis are often the subjects of Rheumatoid Arthritis.

2. *Influences independent of the Individual.*—Everything which causes debility and loss of tone in the extreme circulation, as hæmorrhages from the uterus or elsewhere, deep and prolonged grief, severe

and protracted mental anxiety, acts as a predisposing cause of the disease. It not infrequently results from rapid child-bearing, or too lengthened lactation; also from night-watching. Cold is very frequently an exciting cause of the disease, especially if it has been prolonged, and has caused severe depression of the functions of the nervous system. In one instance I have seen the affection in its most severe form apparently the result of diabetes mellitus.

In some cases injuries or shocks appear to have acted as exciting causes.

Now and then acute rheumatism acts as an exciting cause of Rheumatoid Arthritis.

Malt liquors and wines do not appear to exercise any influence in either causing or protracting Rheumatoid Arthritis.

PATHOLOGY OF RHEUMATOID ARTHRITIS.—The examination of the blood in Rheumatoid Arthritis has failed to discover any constant or pathognomonic changes in that fluid; but as yet the number of analyses has been very limited. One fact of importance they have elicited, namely, the absence of uric acid; and thus we are able to distinguish the blood in this disease from that in true gout.

Pathological anatomy has likewise enabled us to distinguish Rheumatoid Arthritis from gout, on the one hand, and from rheumatism on the other, by demonstrating the absence, in the affected joints, of deposits of urate of soda, which are constant in gout, and by showing the presence of ulceration of the cartilages, and of other structural alterations, which are not found in simple rheumatism, even after repeated attacks.

The consideration of the history and progress of the disease has shown that it differs completely, in its essential nature, from both gout and rheumatism, and fully justifies the rejection of the name "rheumatic gout," which must of necessity convey to every mind the idea of a hybrid disease, a compound of both gout and rheumatism. If a disease should be so designated, because it differs completely in its intimate pathology from both affections included in its compound name, then no fault can be found with the nomenclature.

It seems difficult to persuade those who have been brought up in the old idea of this hybrid affection that such disease has no real existence.

Dr. Aitken, in his excellent work on the Science and Practice of Medicine, although he admits that Hunter warmly opposed this compound appellation, "rheumatic gout," deems it is nevertheless pathologically correct, and thinks that a hybrid disease, depending on the combined cachexia of gout and rheumatism, has a real existence, as recognised by Craigie, Wood, Spencer Wells, and Fuller. On turning, however, to the last-named author, the following passage is met with: "The disease should not be regarded as of a hybrid character, or, in other words, made up in part of rheumatism, in part of gout." And again: "It has no connexion with either of these diseases, beyond that which attaches to it in virtue of its being a constitutional disorder, producing local manifestations in the joints." Might not the very same be said of pyæmia? Is this not a constitutional disease, producing local symptoms in the joints?

It is a much easier task to prove what Rheumatoid Arthritis is not, than to give the slightest clue to what it is: at present I should hesitate to offer a strong opinion as to its nature. It appears to result from a peculiar form of mal-nutrition of the joint textures, an inflammatory action with defective power; but of its dependence upon the presence of any morbid principle, or upon a weakened condition of the vessels or structures of the affected parts, no evidence exists upon which any reliance can be placed. Thus much only appears to be made out: it usually occurs in weakened subjects, and exposure to cold is in many cases the exciting cause of its development.

A full and searching investigation into the nature of Rheumatoid Arthritis is still a desideratum.

DIAGNOSIS OF RHEUMATOID ARTHRITIS.—Perhaps there is scarcely a subject of greater importance in the whole range of joint affections, than the diagnosis of Rheumatoid Arthritis; for upon a correct understanding of it depends the future comfort and physical well-being of a large class of persons.

Rheumatoid Arthritis, as has been already shown, assumes various forms, and individual cases of the disease are often with difficulty distinguished. The affections with which it is apt to be confounded are gout and rheumatism. It is therefore important to be able at once to distinguish Rheumatoid Arthritis from the above-named diseases. Haygarth thus describes the difference between what he termed nodosity of the joints (Rheumatoid Arthritis) and gout, &c.:—

"The nodes appear most nearly to resemble gout: both of them are attended with pain and swelling of the joints, but they differ essentially in many distinguishable circumstances. In gout the skin and other integuments are generally inflamed, with pain, which is very acute, soreness to the touch, redness and swelling of the soft parts, but in no respects like the hardness of bone. The gout attacks the patient in paroxysms of a few days, weeks, or months, and has complete intermissions, at first for years, but afterwards for shorter periods. The gout attacks men much more frequently than women. There is one distressful circumstance which distinguishes this disorder: it has no intermission, and but slight remissions, for during the remainder of the patient's life the nodes gradually enlarge, impeding more and more the motion of the limb; the malady spreads to other joints, without leaving or producing any alleviation in those which had been previously attacked."

The following considerations will enable us to effect the diagnosis in at least the majority of cases:—

It is questionable whether Rheumatoid Arthritis is in any marked degree capable of being inherited; whereas gout is distinctly hereditary.

The sex of the patient does not aid us much. Both sexes are liable to the disease, and the difference in their liability is too slight to enable us to give it much weight in diagnosis. Nor does the age of the patient argue much, as it has been shown that Rheumatoid Arthritis attacks both children and very aged persons.

In many instances Rheumatoid Arthritis is preceded by a condition of ill-health; there are evidences of bad nutrition and exhaustion of the nervous system.

Rheumatoid Arthritis usually begins as a sub-acute disease, and the joint affection gradually increases; but occasionally it commences in an acute form. These latter cases may be mistaken for acute gout or rheumatic fever. There are, however, peculiarities in its course by which it can generally be distinguished from either of these diseases; the most marked being its progressive character, which has been fully described above.

From acute gout it may be distinguished by the length of the paroxysm, the absence of periodicity, by the large and small joints being equally attacked at the outset, and the great toes not being specially involved.

From rheumatic fever or acute rheumatism, by the comparative freedom from constitutional disturbance, the longer duration of the paroxysm, and the absence of acute cardiac inflammation.

From chronic rheumatism it can be distinguished by the comparative absence of structural alteration in the former disease.

The most frequent difficulty which occurs is to separate chronic Rheumatoid Arthritis from chronic gout, and, on referring to the article "Gout" in the present volume, a case illustrative of this difficulty will be seen. There should always be a careful search made for evidences of urate deposits on the ear and at the tips of the fingers, and in the bursæ over the olecranon process of the elbows: but from mere inspection it is difficult to make a diagnosis, especially if the affection has become chronic.

When the diagnosis is very obscure, notwithstanding that the above rules have been followed, it may be at once cleared up by an examination of the blood; in Rheumatoid Arthritis there is no uric acid present: there is also, in most cases, the absence of any material alteration in the urine.

One disease is with difficulty separated from the acute forms of Rheumatoid Arthritis—namely, the joint affection arising from urethral suppuration: the history of the case will of course enable us to clear up the diagnosis.

PROGNOSIS OF RHEUMATOID ARTHRITIS.—Rheumatoid Arthritis is unquestionably a very intractable disease; and this is not to be wondered at, if we consider its ordinary antecedents. Its most common predisposing cause is a thoroughly impaired condition of the system.

and this in many instances has arisen from influences which have been for years in operation: it cannot, therefore, be even reasonably hoped that an affection occurring under such circumstances can be rapidly cured: it is often a great achievement to arrest its further progress.

If the disease is far advanced, and the joints severely injured, it is impossible to restore the articulations to their former state, even if the constitutional tendency to the disease is thoroughly arrested. Still, under such circumstances, it sometimes happens that patients, who for years have been unable to move, will regain their power of locomotion, a result caused by the formation of a kind of false joint in some of the more important articulations. When the disease is less advanced, when the affected joints are few in number, and their mobility but partially interfered with; when at the same time there is freedom from any disease which must of necessity keep up the impaired state of health; lastly, when the progress of the affection has not been rapid,—then a more favourable view may be taken of the case, and a recovery more or less complete may be hoped for.

When the disease is at its commencement, or at least when but little damage has ensued, and at the same time the affection shows little tendency to assume an acute character, then a complete recovery may be anticipated, if proper measures be adopted: if, however, the patient is in any way weakened, or treated as if he were suffering from a sthenic malady, then the probability of the disease becoming deeply engrafted into the system, and causing serious mischief, is greatly increased.

The accurate diagnosis and proper treatment of cases of Rheumatoid Arthritis exercise a very important influence upon its prognosis.

TREATMENT OF RHEUMATOID ARTHRITIS.—The treatment of Rheumatoid Arthritis is a subject of the highest importance, more especially in the earlier stages of the disease, when as yet the joints are not seriously or irremediably injured.

From what has been stated under the "Causes" and "Pathology" of Rheumatoid Arthritis, it will be naturally inferred that a prolonged sustaining plan of treatment is imperatively called for, and that all depleting measures must tend most materially to increase the rapidity and severity of the disease. I have witnessed a great number of cases in which depletion has been persevered in for a time, and with the effect of producing lamentable results, as the joints became perfectly disorganised; and in many of these it is probable an opposite treatment would have been attended with different results.

We must remember that, during the more acute inflammatory stages, the disease must not be treated in the manner recommended to be adopted in gout. Colchicum is always worse than useless, and the patient should be well sustained throughout the whole of the treatment; neither are the alkalies, so serviceable in rheumatism, of any permanent value in Rheumatoid Arthritis.

With the exception of stating that a supporting plan must be adopted from the first, no rules applicable to all cases can be laid down, a fact which must be evident, if we consider the different causes which lead to a condition of habit favourable to the development of the disease. If the disease has been caused by hæmorrhage of any kind, and anæmia exists, then the first object must be to restore the blood to its normal state, and preparations of iron are imperatively called for. If the mere hæmatinic influence is required, the reduced iron (ferrum reductum) may be administered, or the ammonio-citrate or some other very mild salt of this metal; if, however, the anæmia is combined with a relaxed state of habit, the more astringent preparations should be employed, as the sulphate or perchloride of iron. If anæmia exists from other causes than hæmorrhage, similar remedies may be had recourse to.

Ferruginous salts are also of much value in keeping up the power of the heart, but in all cases they should be given in small doses, and persevered in for a long time.

In certain conditions where the nutrition is imperfect from causes often unable to be defined, cod-liver oil may be given with great advantage: if the habit is materially improved by it, the progress of the joint affection is usually checked. Cod-liver oil is particularly indicated in patients of spare habit, and when the disease has been attended with wasting of the body. If the nervous system has been seriously implicated by depressing causes, as anxiety, grief, prolonged attendance on sickness, then remedies more directly affecting this system must be employed, either by themselves, or in conjunction with those already mentioned. Quinine, as a nervine tonic, is often of much service, as likewise, when astringents are indicated, the preparations of cinchona bark; assafœtida, valerian, and ammonia are of value when there is much mental depression.

If the circulation is very languid, guaiacum may be administered with benefit, or guaiacum united with yellow bark; as, for example, the ammoniated tincture of the former medicine, and the simple tincture of the latter.

Guaiacum appears to exert a marked effect upon the capillary circulation, increasing the warmth of the extremities, and rendering the functions of the skin more active.

Arsenical preparations are in some cases of considerable value: how they act is a subject of considerable difficulty to explain. We have clinical evidence demonstrating their peculiar action upon the skin, and it may be that their influence is exerted also upon the fibrous and cartilaginous structures, or they may more especially affect the nervous system as tonics, and thus prove of service. Arsenic may be given either in the form of the arsenite of potash (liquor arsenicalis), or the arseniate of soda (liquor sodæ arseniatis). From repeated trials, I have come to the conclusion that arsenic, in the state of arsenic acid, is less irritating than when in the lower condition of oxidation, and can be given in larger doses.

Iodides are of service in some cases, especially during the more acute stages, or when warmth has a marked effect in causing an augmentation of the pain. Iodide of potassium may be administered alone, or in combination with guaiacum or guaiacum and bark; or the iodide of iron may be given, especially if ferruginous preparations are indicated. The syrup of iodide of iron, in doses of from twenty to thirty drops twice a day, and continued for some months, has in several cases proved of much benefit, even to the extent of completely arresting the progress of the disease.

In special cases special remedies must be employed. If, for example, menorrhagia has been in any way the cause of the debility, and still continues, bromide of potassium may be advantageously had recourse to; and should the muscular system have been wasted, especially the muscles of the affected limbs, nux vomica or strychnine can be given to restore their function.

Mineral waters are often resorted to, from a mistaken view of the nature of the malady. I have seen much injury resulting from their employment, from the debility they have induced. The springs most adapted for the subjects of Rheumatoid Arthritis are those of Schwalbach, Pyrmont, and Spa, or any other ferruginous waters, which sit easily upon the stomach. The stronger saline and alkaline waters, as those of Carlsbad, Wiesbaden, and Vichy, unless used with great care, are apt to aggravate the disease. In some very chronic cases, the springs of Wildbad and Gastein have proved of much service.

Change of air, occupation, and scenery, by aiding the general health, exerts a beneficial influence upon the progress of the malady.

Local Treatment of Rheumatoid Arthritis.—In the early stages, when there is tenderness and swelling of any joint, relief is often experienced from the application of blisters, and a most convenient form is the cantharides liniment of the British Pharmacopœia: it produces, in almost all cases, full vesication, and with little annoyance; it can be frequently applied, so as to produce a series of flying blisters. Under this treatment the effusion will often quickly subside, and the tenderness become much lessened or even removed, and the liability to serious or permanent injury of the joint is thereby greatly lessened. Other forms of counter-irritation, as iodine paint, croton oil, may be used, but I believe the blister treatment above mentioned is productive of more benefit.

When the affection of any joint has become more chronic, and blisters have effected all they are able to accomplish, further benefit may be obtained from the use of plasters, which act not only by the slight counter-irritation they produce, but likewise, if properly applied, from the support they afford to the joint. The kind of plaster selected should depend on the requirements of the case. If little more than simple support be needed, soap plaster may be used, and the joint may be bandaged with narrow strips of this, spread by machine on linen. If more irritating applications are desired, then the galbanum or ammoniacal plaster can be used, or the ammoniacal and mercurial, or the iodine plaster: when using the two latter, severe irritation of the skin should be avoided.

The application of lotions containing spirit of such a strength as to cause but slight irritation of the skin sometimes gives great relief. If there is much pain, the belladonna liniment may be used in lieu of the simple spirit.

Baths are in some cases useful, especially when the function of the skin is defective; but care must always be taken that debility be not induced, otherwise any good result is more than counteracted.

Bathing is certainly not curative in this disease, it can only be looked upon as palliative, and should be employed with this understanding.

In chronic cases, when friction can be used without increasing the tenderness of the parts, it may be employed either alone, or combined with some stimulating liniment; or the joint may be well sponged with strong brine, and then rubbed dry, so as to cause the salt to enter the skin.

It is often asked, whether it is desirable to allow movement of the affected joints? I believe the best answer to be this: never allow such an amount of movement as will cause the joint to be more painful on the following day; but any motion short of this may be employed with advantage. If this rule were followed, it would prevent the use of joints recently or acutely attacked, and rest in such cases aids the subsidence of the inflammation; on the other hand, it would allow those articulations in which the disease had become chronic to be moved to such an extent as to stay the wasting of the muscles of the limb, and to prevent the stiffening of the joints.

DIET AND REGIMEN IN RHEUMATOID ARTHRITIS.—As far as the fluid portion of the diet is concerned, it is a matter of indifference whether malt liquors, wines, or distilled spirits are taken, provided that they agree with the stomach; it is far different in true gout.

Whichever kind of alcoholic beverage causes the patient to eat with most relish, and digest with most comfort, should be selected. There is no necessity to give enough to stimulate, but only a sufficient amount to sustain the vigour of the assimilating functions. I am convinced that wines and malt liquors have no influence in favouring the development of Rheumatoid Arthritis, although they powerfully predispose to the production of gout. The solid portion of the diet should be of the most nutritious character and of easy digestion, for our main object is to keep up the strength of the system; nor have we any proof that an excess of animal food tends to increase the disease, as is the case in gout. Meat should form a good proportion of the diet, if the stomach is capable of digesting it; and whatever articles of diet are found to improve the general health may be given with advantage.

A frequent change of air and scenery should be advocated, all prolonged mental exertion avoided, and, as far as possible, all causes of anxiety should be removed.

A residence in a moderately warm country during the winter months is desirable, but the air of the place should be dry and bracing. Such a climate will enable the patient to take exercise and have fresh air when otherwise he would be confined to the house.

If the joints allow of it, moderate exercise should be enjoined, but not sufficient to cause subsequent fatigue.

The clothing should be warm, but much perspiration avoided.

RHEUMATISM.

By Alfred Baring Garrod, M.D. F.R.S.

The subject of Rheumatism will be discussed in the present article under the heads of Articular and Muscular Rheumatism; the first, or Articular Rheumatism, in its more acute form, being commonly known as Rheumatic Fever. Such a division has been adopted, as it is a question, at the present day, whether the articular and muscular affections are in reality manifestations of the same disease.

A. Articular Rheumatism.

Definition.—(1) Acute Articular Rheumatism. A specific inflammation of the structures in and around the joints, attended with great febrile disturbance; erratic; not accompanied with deposits of urate of soda, and (?) not leading to suppuration. (2) Sub-acute Articular Rheumatism. The same affection as the above, but manifested in a much less intense degree, and with little febrile disturbance; generally following upon the acute disease.

Synonyms.—Rheumatic Fever; Arthritis.

History.—Although there is every probability that mankind was afflicted with Rheumatism from the very earliest periods, still we gather from the writings of ancient physicians that they did not separate this malady from other forms of joint disease, but comprehended all of them under the general term "arthritis," the only distinction being made dependent on the particular articulations implicated.

Monsieur Baillon, a French physician, first made use of the word Rheumatism (from *ῥεῦμα*, a stream, a fluxion): in a Latin treatise published in 1642, entitled "De Rheumatismo et Pleuritide Dorsali," he separates this disease from gout in the following sentence: "Gout is a disease of a certain part, and periodical; Rheumatism of the whole body, and more uncertain in its attack."

Sydenham afterwards makes a distinction between the two affections: "This disease, when unattended with fever, is frequently mistaken for gout, although it differs essentially therefrom, as will easily appear to those who are thoroughly acquainted with both diseases; and hence it is, perhaps, that physical authors have not mentioned it, unless indeed we esteem it a new disease." Cullen thus defines Rheumatism: "A disease from an external and often an evident cause; pyrexia; pain about the joints, following the course of the muscles, fixing upon the knees and larger joints in preference to those of the feet and hands—increased by external heat."

Even since Cullen's time there have been authors, both in this country and abroad, who have included Rheumatism and gout under the same category, amongst whom stands prominently the name of M. Chomel.

The late Sir Benjamin Brodie, in his work on diseases of the joints, clearly separates Rheumatism from rheumatoid arthritis, although he designates the latter affection ulceration of the cartilages.

At the present time it may be confidently asserted that, omitting purulent affections of the joints and those connected with urethral suppuration, there exist at least three well-marked articular diseases dependent on morbid states of the system; these are Rheumatism, gout, and rheumatoid arthritis: and probably to them we may add a fourth, namely, muscular rheumatism.

Description of Acute and Sub-acute Rheumatism.—To illustrate this subject we will give a sketch of an acute attack of Rheumatism, such as is daily met with in practice. A young woman, possibly somewhat out of health, is exposed to severe cold, or to cold and damp conjoined; she feels a distinct chill or rigor, and this is followed, probably during the second or third day from the exposure, by the development of the joint affection; the ankles become painful and unable to bear the weight of the body, and on examination they are found tender to pressure, swollen, unduly hot, with a distinct flush upon the surface; at the same time the system exhibits a state of febrile excitement, the pulse is rapid and commonly hard, the whole surface hot and bathed in perspiration, having a peculiar acid, at least acrid, odour. The tongue is coated with a thick creamy fur; there is loss of appetite, but increased thirst and a constipated state of the bowels; the urine is usually scanty and high-coloured, and gives rise on cooling to a copious red deposit. The inflammation is seldom confined to one joint, but gradually extends over the whole body. The larger joints are more frequently affected than the smaller in the earlier periods of the attack.

The erratic nature of the affection is usually well exhibited; at one time the knees and ankles, at another the elbows and wrists suffer; and not infrequently the development of inflammation in one set of joints is accompanied by its rapid subsidence in another.

A symmetry is often shown in the order of attack: the right ankle, then the left; the right knee, then the left; and so on for the other articulations. It is a matter of astonishment to observe how quickly and completely the inflammation will subside in any part. A knee, for example, will one day be intensely hot and swollen, and so exquisitely tender as not to bear the weight of the bed-clothes; but on the following day will scarcely show any evidence of its previous suffering. This condition of the patient may continue for many days or even weeks, the duration depending partly on the intensity of the disease, partly on the habit of the patient, and partly on the treatment adopted. Its usual duration, when under no special treatment, is from ten days to three or four weeks; but perhaps no disease exhibits greater differences in this respect, and hence the difficulty, except when a number of cases are compared, of accurately estimating the value of the various plans of treatment which have been employed for its cure. Cases are now and then met with which spontaneously terminate in five or six days, others which run a course of six or even eight weeks. With few exceptions, the pain of the joints and the febrile disturbance are greater at night than in the day; but the nocturnal exacerbations are less marked in rheumatic fever than in acute gout.

After a varying time the joints become free from redness, swelling, and tenderness, the pulse lowered to its healthy standard, the temperature and sweating of the surface diminished; at the same time the tongue cleans, the thirst abates, the appetite returns, the urine clears, and in short the system is restored to its healthy state, with the exception of a certain amount of wasting and debility, necessarily induced by the increased wear of tissue and the defective nourishment which have taken place during the period of febrile disturbance. From these, however, the patient soon recovers, when able to take a generous diet and enjoy fresh air and exercise.

It is not unusual to find that patients after recovering from attacks of rheumatic fever, as far as regards the constitutional symptoms, are liable to suffer from pains in those joints which have been the seat of inflammation: these pains are not constant, but assume a neuralgic character, and will generally be found to be in proportion to the length of time the joints were affected during the febrile attack.

Thus far our description has been confined to a case of acute rheumatism, in which the inflammation has been limited to the joint structures: unfortunately, however, it is not always so, for in a large percentage of cases the covering or lining membrane of the heart, or even the substance of the organ, becomes implicated, and peri- and endocarditis ensue. Sometimes the heart disease follows a somewhat sudden subsidence of the joint inflammation, but more commonly the articular and cardiac affections run a simultaneous course.

When inflammation of the pericardium takes place, the patient usually experiences a sensation of tightness or pain in the chest, but not always, as the acute discomfort caused by the condition of the joints draws the attention altogether from the slight uneasiness due to the cardiac complication. On applying the stethoscope a distinct friction or rubbing sound is heard, often limited at first to a small extent of surface, generally at the base of the heart, but it commonly spreads over nearly the whole of the surface of the organ; when the amount of effused fluid is large, there is increased dulness of the percussion note over the cardiac region, and the heart's sounds are diminished in intensity, and heard as if from a distance. When, on the other hand, the endocardium is implicated, abnormal heart sounds are produced usually at the aortic or mitral valves, giving rise to basic or apex systolic murmurs; or, if the closure of the aortic valve becomes imperfect, a basic diastolic murmur is also developed. These various phenomena are due to the pouring out of lymph and serum upon the surfaces of the pericardium, or the secretion of plastic lymph or fibrinous coagula upon the valves of the heart. When the cardiac substance is involved, great irregularity and extreme feebleness of the action of the heart ensue, often leading to sudden and fatal collapse. For further details concerning the physical signs and symptoms which occur in pericarditis and endocarditis, the reader is referred to the articles devoted to those diseases, as these affections, when they arise from rheumatic inflammation, follow the same course and exhibit the same phenomena as when due to other causes.

Pleurisy of a true rheumatic nature sometimes occurs during the progress of rheumatic fever, and the phenomena are the same as in the ordinary forms of acute pleuritis.

More rarely the peritoneum becomes affected by rheumatic inflammation.

When the heart is implicated, delirium is usually present, especially at night; but in some few cases the rheumatic condition appears to attack the membranes of the brain, and then the symptoms of cerebral meningitis are produced. Sometimes the membranes of the spine are involved, and symptoms of spinal meningitis are set up.

Chorea is apt to supervene in young subjects after the cessation of the febrile disturbance in rheumatic fever, where the heart has been implicated.

Description of Sub-acute Articular Rheumatism.—At times, from various causes, as the nature of the patient's constitution or the presence of cardiac complication, articular rheumatism assumes a form to which the name sub-acute may be properly applied; the joint symptoms remain, but in a much less severe degree, and there is an almost complete absence of febrile disturbance. Such a condition may continue for weeks or even months, at one time relieved, at another aggravated, and the disease may then be compared to a similar sub-acute form not infrequently seen in gout, and to which the term chronic is applied.

In sub-acute rheumatism there is often some tenderness of the joints, slight swelling, and heat, but the disease differs from both chronic gout and rheumatoid arthritis, inasmuch as it may continue for a long time without leading to any great deformity or permanent injury to the articulations.

It will be seen that the statements made above are at variance with those commonly met with in works on this disease, in which the results ascribed to the sub-acute affection are so formidable. The difference is easily explained: most authors attribute to chronic rheumatism the changes that in the present work are described under the head of rheumatoid arthritis.

The occurrence of one attack of rheumatic fever imparts a great susceptibility to the system for its return, which a second augments, and thus after a time the patient is liable to become the victim of frequent seizures, though these will probably assume a less sthenic form, and are often of such a character as to entitle them to be called sub-acute rather than acute. When a patient has been brought to this condition, the most trifling exposure to cold, or even the slightest depressing cause, may prove sufficient to light up the disorder.

Even in individuals who have suffered long and severely from repeated attacks, it is unusual to find any very serious alterations produced in the affected joints; they may for a time remain more or less swollen from effusion of fluid, the ligaments may become stretched and relaxed, but there is no permanent thickening left either from the deposits of the chalk-like matter (urate of soda) so constantly found in gout, or from the formation of false cartilages and other changes, the effect of rheumatoid arthritis.

Consideration of the different Phenomena in Acute Rheumatism.—The various symptoms which are observed in cases of acute rheumatism demand further investigation than could be given them in the above sketch, and the first which claim attention refer more especially to the joints themselves. The pain is generally very severe, but less intense than in gout; the swelling also is usually less marked than in the latter disease; there is rarely found extreme tension of the skin, and seldom œdema or desquamation of the cuticle; the redness is likewise less in Rheumatism than in gout, although this symptom may sometimes be present in a marked degree; and, lastly, the enlargement of the veins leading from the inflamed joint is certainly much less prominent in this disease than in true gout.

Rheumatic inflammation has a decidedly erratic disposition, and, as it were, flies from joint to joint, or at last one articulation suddenly ceases to exhibit inflammatory symptoms, and another, probably the corresponding articulation, becomes as suddenly implicated; and this alternation may occur many times during a single attack. A certain amount of symmetrical action is likewise observed, as in almost all diseases the symptoms of which depend on an altered condition of the blood. On reflection it will be found that our surprise should rather be excited by a want of symmetry than by its occurrence; for supposing that any morbid element has a peculiar attraction to any one joint—a knee, for example—it is necessarily drawn to the second knee more powerfully than to any other joint; or supposing again that the circumstances in one particular joint more especially favour the development of any specific form of inflammation in it, the conditions of the corresponding joint on the other side of the body must do so likewise.

It has been stated that the swelling of the inflamed joints is less in Rheumatism than in gout, and the same holds good with the pitting on pressure, and the subsequent desquamation of the cuticle. This last symptom, so constant in gout, is not observed after rheumatic inflammation: but it must not be concluded that because pitting is present the inflammation is not rheumatic; this would be erroneous, for cases of genuine rheumatic fever are now and then met with in which pitting of the skin is well marked: when this phenomenon occurs, it is generally in weak subjects. In several such instances I have proved, not only by carefully examining all the symptoms, but also by analysing the blood, that the cases were of a true rheumatic character, and had no relation to gout.

The constitutional symptoms which are most prominent in acute articular rheumatism next require consideration. The first of these, one of great importance, is the temperature of the body. That there is a well-marked heat of surface is evident to the touch, but the indications of the thermometer are far more trustworthy; for they show correctly the amount of tissue-waste going on in the system, and cannot be be masked by the influence of the mind or other circumstances which render some of the subjective symptoms of comparatively little value.

Dr. Sidney Ringer, whose researches on temperature in disease are of much value and interest, has made observations on three cases of acute rheumatism which were communicated to Dr. Aitken, and published in his work on the Science and Practice of Medicine. In some fatal cases, complicated with severe pericarditis, the temperature rose as high as 106° Fahr., 109° Fahr., and 110° Fahr. before death.

In some instances of acute articular rheumatism I noticed the temperature, and found it to vary from 100° Fahr. to 104° Fahr.; but these observations have been made in cases in which recovery has taken place. The pulse, as already noticed, is usually quiet in acute rheumatism; it is also hard and full; but exceptional cases are now and then met with in which the frequency is never great, although the febrile disturbance and temperature run high. More reliance can be placed upon the heat of the body than upon the rapidity of the heart's action in all cases of acute inflammation; and this especially holds good in Rheumatism, as the cardiac complications so frequently present in this disease alter the character of the pulse, independently of the amount of tissue-change which is going on in the system.

The perspiration is generally considered to be intensely acid in acute rheumatism; in several cases I have found it less acid than in healthy subjects; but it must be remembered that the amount of perspiration is excessive. The peculiarity of the skin secretion in this disease depends on its acridity perhaps more than on excessive acidity.

Condition of the Blood in Acute Articular Rheumatism.—In acute rheumatism, if blood is drawn from a vein, the clot is found to exhibit a buffy coat, and is frequently cupped—appearances which indicate that the fibrin is increased in quantity, and that this principle is also probably somewhat altered in quality.

Many discrepant statements have been made upon this subject: according to Haller, the blood yields a thick and firm clot in this disease, but others assert that under the buffy coat the clot is found to be loose and friable;—probably both are correct in different instances.

Andral and Gavarret analysed the blood in fourteen cases of rheumatic fever, and found the maximum amount of fibrin to be 10·2 parts in the 1000; the minimum 2·8 parts; the mean was 6·7: healthy blood yields, according to these observers, three parts per thousand.

The serum of the blood does not appear to be very sensibly altered in composition; Andral and Gavarret found an increase in the solid residue left by evaporating this fluid, the mean amount being 86·0 per thousand against 80·0 per thousand in healthy blood.

The same chemists found a diminution in the total solids: thus the mean in the above-mentioned fourteen cases was 194·6 parts of solid matter to 805·4 parts of water in the 1000 parts of blood; whereas in healthy blood the ratio between the solid residue and water is 200·0 to 790·0.

In several analyses which I have made of the blood in acute rheumatism, I have found the amount of fibrin notably increased—namely, from four to six parts in the 1000—and the clot has usually been buffed and cupped. The serum in thirty-five cases was found distinctly alkaline in reaction, it presented a healthy appearance, its specific gravity was somewhat less than in health, and in no case was any uric acid detected either by the thread experiment described in the article on Gout, or by the ordinary method of separating this acid from blood. The absence of uric acid or urate of soda is important, as it at once shows essential difference between gout and Rheumatism.

Urea does not exist in the blood in acute rheumatism in quantities larger than in health, except in cases in which the kidneys have been either previously affected, or have become congested during the progress of the disease. As above stated, the serum was always distinctly alkaline in reaction: this fact was particularly observed and noted, as it has been affirmed that the serum is sometimes acid in rheumatic fever. There are, as yet, no recorded observations indicating the amount of the alkalinity. No abnormal principle has been found in the blood; lactic acid has been assumed to exist in it, but no proof has been given of its presence.

In the sub-acute and chronic varieties of Rheumatism no marked alterations are found; the fibrin is increased but slightly, and only in proportion to the amount of the febrile disturbance. In ten cases the maximum of the fibrin was 5·1, the minimum 2·6, and the mean 3·8 parts in the 1000 parts of blood.

Urine in Acute Articular Rheumatism.—Examinations of the urine in acute rheumatism have been frequently made with the following results:—To the eye its appearance varies much in different cases; as a rule it is high-coloured and scanty, clear when first passed, but speedily becomes turbid from the deposition of urates; when copious it sometimes remains bright even after it is cold. These characters are by no means peculiar to rheumatic fever, but are seen in many febrile affections. The deposited urates are often of a deep tint from the colouring matters of the urine being in excess and attracted to the uric acid salts, and if the fever runs very high, and especially if portal congestion is present, the colour becomes bright red or deep pink. On a more minute examination, it is found that the secretion of water by the kidneys is usually diminished; the solids of the urine are increased. The increase in the solid matter is due chiefly to the augmentation of the urea, partly also to an increase of the colouring matter. The uric acid is augmented: Dr. Parkes has found as much as seventeen grains in one case; the largest amount I have obtained has been fifteen grains. It should be remembered that this increase of uric acid in the urine is not peculiar to rheumatic fever; it occurs likewise in other febrile diseases, unless the excreting power of the kidneys is injured. The chlorides are diminished during the febrile disturbance, but less so than in pneumonia. Dr. Parkes found the sulphates much increased in several cases in which the inflammation of the joints and the febrile disturbance were severe. When alkaline remedies are not administered, the acidity of the urine is usually great. Albumen is now and then met with during the course of this disease, sometimes only for a day or two, but its occurrence is much less frequent in rheumatic fever than in pneumonia. No proof has yet been given that lactic acid exists in the urine in rheumatic fever in greater amount than in healthy urine.

Cardiac and other Inflammatory Affections in Acute Rheumatism.—It has been stated that in a large percentage of cases of rheumatic fever the structure of the heart becomes involved, and as such complications are of vital importance to the patient, they deserve in this place further consideration.

It had long been suspected that some relation existed between acute articular rheumatism and inflammatory diseases of the heart. In the first volume of the Medico-Chirurgical Transactions there is a paper by Sir D. Dundas on this subject, in which he states: "In all cases which I have seen, this disease has succeeded one or more attacks of rheumatic fever. In one case, the affection of the heart appeared at the commencement of the rheumatic fever, and its action was so rapid that the pulse could not be counted for many days; much difficulty of breathing and oppression, attended with a sense of debility, took place; and the inflammation, pain, and swelling of the extremities, after having shifted from one joint to another for many weeks, subsided; but the affection of the heart continued, generally attended with great pain, producing in the progress of the disease, and towards its close, a considerable disposition to dropsy, under which the patient lingered for many months."

As far back as 1788, it would seem that Dr. Pitcairn had noticed that persons subject to Rheumatism were attacked more frequently than others with symptoms of heart disease, and he considered that the two diseases often depended on a common cause. The connexion between Rheumatism and cardiac disease was also known to Dr. Wells, Dr. Baillie, and others. The heart affection, however, was regarded by these authors rather as the effect of a metastasis of the rheumatic inflammation from the joints to this organ, than as an essential part of the disease itself. Some French authors claim the merit of the discovery of the close relationship for M. Bouillaud, who in his work, published in 1840, called especial attention to the coincidence of pericarditis, endocarditis, and pleurisy with acute articular rheumatism. Since the above period, the relationship has been fully determined, and these diseases are now looked upon as portions of the rheumatic affection, and not as mere accidental complications.

There is considerable discrepancy of opinion among different authors as to the kind of cases of rheumatic fever in which cardiac inflammation is most likely to occur. Some are of opinion that even in the slightest forms of the disease, when febrile disturbance is moderate, the structures of the heart may become seriously affected. Others think that the liability to the heart disease is far greater in the severe forms of articular rheumatism; and that even when the joints are not much implicated, still the systemic disturbance is always great, and the peculiar phenomena of the disease strongly marked. As far as my own experience goes, it amounts to this, namely, that although the severer forms of the articular disease are very apt to be complicated with cardiac inflammation, yet even in the very slight forms, measured by the febrile and joint symptoms, serious mischief may arise in the heart; and several such cases have come under my own observation. When the heart is much influenced, this will of itself be a source of great constitutional disturbance. It is supposed by some pathologists that the vegetations which take place on the valves of the heart are not always due to endocardial inflammation, but may arise entirely independently of such, and are owing rather to an altered state of the blood itself.

It is a matter of extreme difficulty to determine the relative frequency of the cardiac affection in rheumatic fever; in young subjects the heart is much more prone to be attacked than in adults; the kind of treatment adopted in the early stages appears likewise, from the statistics of Dr. Dickinson, to have a considerable influence upon the result. Bouillaud thought the heart became affected in one-half of the cases, but Dr. Macleod in about one-fifth only.

MORBID ANATOMY OF ARTICULAR RHEUMATISM.—The opportunities of examining joints affected with rheumatic inflammation are not numerous, as the articular affection never kills, and a fatal termination, even when the heart or other important organ is attacked, is fortunately rare. In a few instances, where death has occurred when inflammation of the joints was actually present, I have found considerable redness of the synovial membrane, and increased vascularity of the synovial fringes; an augmentation of the synovial fluid, and sometimes little gelatinous coagula of fibrin; under the microscope nucleated cells were seen, but none of the ordinary appearances of pus. In one case of mono-articular disease, the fluid was indeed turbid with pus cells, but in this instance some doubt existed as to the true nature of the affection.

In Dr. Fuller's work a detailed account is given of the post-mortem appearances in sixteen cases of acute rheumatism in St. George's Hospital. In eight of these cases the joints were examined: in some of them the only morbid appearances were increased vascularity, a thick tenacious fluid, with granular globules or a few pus cells; in some the fluid was turbid, and soft fibrinous coagula were found; in other cases pus is described as being present, both in the joints and likewise along the tendons.

In two cases nothing abnormal was observed: but in one the inflammation had subsided some weeks before death; and in the second, as far as the history is given, the local mischief was not great.

Chomel, who examined a knee-joint two days after the cessation of acute rheumatic inflammation, found an excess of synovial fluid, but no other morbid change.

In examining joints which had been frequently and severely affected in rheumatic fever, but in which the inflammation had completely subsided, the only appearance I have observed, indicating any deviation from the normal condition, has been a lax state of the ligaments of the articulations, and an opacity of the articular cartilages, or a loss of the natural bluish-white opalescence of this tissue.

No mention has been made of ulceration of the articular cartilages, and I have had proof that at least a dozen attacks of rheumatic inflammation may occur in a joint without any such change taking place.

In no case has the slighest trace of urate of soda been found, although very carefully looked for, and on one occasion I had the opportunity of examining and comparing two knee-joints—one that of a young man who had died from heart disease at the time that the joint was suffering from recent rheumatic inflammation; the other, that of a man who had been killed by an accident, which had previously caused the development of gouty inflammation of the part. The difference in the two cases was very striking: the vascularity in each was about the same; in the latter there was the characteristic white thickening of the cartilages from the crystalline deposition, and in the former the complete absence of such an appearance.

For the changes which ensue when rheumatic inflammation attacks the pericardium, endocardium, or substance of the heart, as likewise when the pleura or membranes of the brain are affected, we must refer the reader to the articles on the various diseases of those structures.

CAUSES OF RHEUMATISM.—The causes of acute rheumatism may be divided into (1) those which belong to the individual, and (2) those which are external to and independent of the patient.

(1) *Influences dependent on the Individual.—Hereditary Predisposition.*—Although the influence derived from this cause is not so well marked as in the case of gout, still it is very powerful. Chomel and

Requin stated that, in 72 cases, 36 had rheumatic parents, 24 had healthy parents, and 12 were unable to give any information upon the point. This statement, however, is of little value, from the fact that these physicians did not distinguish cases of Rheumatism from those of gout.

Dr. Fuller traced an hereditary influence among the rheumatic patients admitted into St. George's Hospital, in nearly 29 per cent: the writer has found that in about one-fourth of his patients hereditary predisposition could be traced; many other authors have come to a similar conclusion; and it may be looked upon as an established fact, that a disposition to Rheumatism can be inherited.

Age.—Young people are much more liable to be attacked with rheumatic fever than those more advanced in years, and in this respect Rheumatism differs essentially from gout. Heberden gives an instance in which Rheumatism occurred in a child only four years old. Others have made similar statements. Haygarth states that rheumatic fever affects persons of all ages from five to seventy-two inclusive; more frequently from six to thirty, but most frequently from sixteen to twenty years of age.

Sex.—In a table given by the writer of 51 consecutive cases of rheumatic fever which came under his care, 31 were females, and 20 were males. There are, however, many discrepant statements with regard to this point. From many statistics it would appear that men are more subject to Rheumatism than women: thus it was found that out of 289 cases of acute rheumatism admitted into St. George's Hospital, 151 were males, and 138 were females; and again, out of 136 cases of the same disease recorded by Dr. Latham, 75 were males, and 61 females. In Dr. Haygarth's cases, 99 were males, and 71 were females.

It has been thought that women, after the catamenial period, are as liable to be attacked with Rheumatism as men. I have no hesitation in saying, that if this remark applies to true articular rheumatism, it has little or no value; for after the age of forty-eight it is very rare to find either men or women affected for the first time with this disease. Women, however, are prone to have both gout and rheumatoid arthritis upon the cessation of the menstrual function.

State of Health.—There can be little doubt that articular rheumatism is more apt to become developed in individuals who have from some cause or other become weakened, either from insufficient nourishment or from disease: hence one cause of its more frequent occurrence amongst the poor and ill-fed than amongst the rich. It is very common to meet with it in women who are suckling, the debility arising from over-lactation giving them greater liability to be attacked.

(2) *Influences external to the Individual.*—Cold is certainly a very common exciting cause of articular rheumatism, and on referring to tables which have been constructed by different observers it will be found that this cause is far more potent than any other. The majority

of patients are able to trace their malady to some one direct exposure to cold, and especially to cold combined with moisture. The more liable the person is from causes within himself, the less is the exposure which is necessary to develop the disease; a cold draught when over-heated, a damp bed, getting thoroughly wet through and becoming chilled, are the circumstances most prone to excite articular rheumatism. It would appear probable that cold acts by causing a sudden check to the function of the skin.

Scarlatina is often followed by an articular affection which has all the characteristics of acute or sub-acute rheumatism, but which must be separated from the fearful purulent disease of the joints occasionally met with under these circumstances. If the Rheumatism which follows scarlatina is of the same character as rheumatic fever, its occurrence may be explained by the fact, that the scarlatina poison acts especially upon the skin, and greatly influences the cutaneous function, which is proved by the subsequent severe desquamation of the cuticle.

It has been supposed that other causes, as the suppression of the menstrual discharge, or of habitual fluxes of various kinds, can act as the exciting cause of articular rheumatism; but if the recorded instances are carefully inquired into, it will be found that they are either cases of gout, or of some kind of purulent joint affection.

In a certain number of cases of rheumatic fever, the patients are unable to state any exciting cause of the attack, and if the predisposition to the disease is very great, it may often occur without any appreciable cause.

Effects of Climate, Seasons, and Weather.—Dr. Aitken gives, in his work on Medicine, the returns, by Colonel Sir A. Tulloch, of Rheumatism in the regiments stationed at home and in the different colonies belonging to this country. Out of 1000 soldiers admitted at the military hospitals, in Jamaica, 29; Nova Scotia and New Brunswick, 30; Bermuda, 33; Malta, 34; Ionian Islands, 34½; Gibraltar, 38; Canada, 40; Mauritius, 46; Windward and Leeward Command, 49; United Kingdom, 50; and Cape of Good Hope, 57, were sufferers from Rheumatism. The following passage from the above work contains all that is known on this subject:—"It is not, therefore, in the coldest climate that Rheumatism is most prevalent, but in those seasons and in those climates remarkable for damp and variable weather; and thus, says Sir A. Tulloch, 'we find in the mild and equitable climate of the Mediterranean or the Mauritius the proportion of the rheumatic affections even greater than in the inclement regions of Nova Scotia and Canada; and though some of the provinces of the Cape of Good Hope have been without rain for several years, yet Rheumatism is more frequent in that command than in the West Indies, where the condition of the atmosphere is as remarkably the reverse.' Exposure to heat, however, would appear to have much influence in the production of Rheumatism; for we find the returns of the navy show a considerably larger proportionate number of attacks than those of the army—the number per thousand annual mean strength attacked in the Mediterranean fleet being 63·9, in the West Indies and North American station 69, and in the South American Station 72·3."

Chomel remarks that Rheumatism is seldom met with near either the equator or the poles, but that it becomes more frequent as we proceed from these regions, and that it is more especially rife in the temperate zones.

In England the occurrence of rheumatic fever appears to be much more common in the eastern than in the western counties: this is usually accounted for by the exposure of the former portion of the country to the cold north-east winds.

As far as seasons are concerned, it may be stated that no part of the year is exempt. Haygarth's tables give the following results in 150 cases:—The disease began in January in 21; February, 11; March, 9; April, 18; May, 11; June, 11; July, 14; August, 5; September, 8; October, 13; November, 13; December, 16.

It will be found, however, that in different years the relative number for each month is liable to considerable variation.

PATHOLOGY OF RHEUMATISM.—The name Rheumatism implies that the disease has been considered to be dependent upon some altered condition of the blood, and the ancients, confusing Rheumatism with gout, necessarily regarded it as having the same humoral origin.

One set of pathologists look upon Rheumatism simply as an inflammation of certain fibrous and serous membranes. This was the view held by Cullen, who remarks that, although some have attributed the disease to the existence of a peculiar acrimony in the system, it was without reason.

Sir C. Scudamore held the same views as Cullen, and makes the following remark:—"It may be stated, that the predisposition to Rheumatism consists in a deficiency of healthy tone in textures connected with joints and muscles, and in nerves, so as to be affected in this peculiar manner by the influence of variable temperature. If we lose sight of the humoral term Rheumatism, we shall come to the simple fact, that, in a condition of susceptibility, cold, or sudden reduction of temperature, makes a particular impression on the vessels and nerves near the surface, and produces a painful affection of certain textures, which is attended with more or less inflammation, the phenomena of which are so far of a peculiar nature, that we either consider the disease specific, inasmuch as the symptoms differ in their constituent character from those produced by other inflammations, or we may view the effect in the light of common inflammation, modified on the one hand by the nature of the exciting cause—the external one, cold,—and on the other hand by the particular species of textures which become affected."

With slight modifications, quite unnecessary to detail, this view resembles that of some authors of the present day who look upon

Rheumatism as unconnected with any essential morbid state of the animal fluids.

Similar objections to those which have been raised against Cullen's views of gout apply to the above attempted explanation of the pathology of Rheumatism.

A second set of pathologists consider the phenomena of Rheumatism as produced by the presence of some peculiar principle in the blood, either one altogether foreign to its healthy constitution, or an augmentation of some normal ingredient.

Dr. Prout first threw out the idea that lactic acid was the principle in question, and this view has found many supporters. It has been argued that the skin is the great eliminator of lactic acid; that cold is the chief exciting cause of Rheumatism; and that, perhaps, bad or insufficient nourishment may lead to an increased formation of this acid, and cold to its defective excretion, and the combined influence may cause a large accumulation in the blood. This explanation of the disease has much in it which is probable, but at present it is devoid of anything like absolute proof. The only observations which appear to give direct support to it are those of Dr. W. B. Richardson, who has shown that when lactic acid is injected into the peritoneal cavity of dogs, it causes both peri- and endocarditis. The post-mortem appearances in different animals exhibited a red, thickened, and œdematous state of the mitral valve, with fibrinous beadlike deposits on its surface; the endocardium lining the left ventricle was intensely vascular, resembling bright-red velvet; and the aortic valve was of a deep-red colour, its fine borders being thickened and everted; the pericardium was red and injected with effused lymph upon the surface of the ventricle. In some cases the tricuspid valve exhibited a similar injected condition. No swelling of the joints was observed during the life of the animals, and no morbid appearances exhibited after death.

These experiments are instructive, but they only prove that the absorption of a large quantity of a free acid by the peritoneum leads to inflammation of other surfaces of a similar nature; and it has yet to be shown whether like phenomena would not be induced by the absorption of other acid or acrid substances. If the injection of other acids, as acetic and formic acid, should produce the same results, there would be no proof that Rheumatism has any relation to lactic acid.

The pathology of articular rheumatism must be allowed to be in a very unsettled state, and further observation and experiments are required before we can arrive at any satisfactory conclusion with regard to it.

Diagnosis.—It will be necessary under this heading to consider the salient points of difference which exist between Rheumatism and the diseases with which it is liable to be confounded.

The affections for which acute rheumatism is likely to be mistaken are acute general gout, acute rheumatoid arthritis, pyæmia affecting the joints, and the acute forms of urethral rheumatism.

From acute general gout it may be separated by the following peculiarities :—

The absence of uric acid in the blood.

The prevalence of extreme sweating, and the acrid odour of the perspiration.

The great amount of constitutional disturbance compared with the joint affection.

The tendency to acute cardiac inflammation.

From acute rheumatoid arthritis, by the great constitutional disturbance, the acrid sweats, the liability to cardiac inflammation, and the erratic nature of the joint disease.

From pyæmia, by the erratic nature of the inflammation, by the great amount of perspiration, by the liability to cardiac inflammation, and by the absence of the disorganization of the joints produced by the latter disease.

From acute inflammation dependent on urethral suppuration, by ascertaining the history of the case, the greater constitutional disturbance, the more general character of the joint affection, and the absence of severe disorganization of the articulations.

The sub-acute form of articular rheumatism may usually be distinguished from chronic gout and rheumatoid arthritis by attention to the above differences and a careful inquiry into the history of each case.

PROGNOSIS OF ARTICULAR RHEUMATISM.—Articular rheumatism, even in its most acute form, though extremely painful, can scarcely be looked upon as a very fatal malady, but it may lay the foundation of the most serious disease of the heart. Dr. Aitken states that the deaths from rheumatic fever hardly exceed one out of every thousand cases of death from all causes, but this applies only to those immediately resulting from the attack; it is almost impossible to arrive at anything like a correct estimate of the proportion who die from its after-effects. When rheumatic fever proves fatal, it is either from acute disease of the heart, probably involving the substance of the walls of the organ, and leading to sudden collapse, or, much more rarely, death may result from acute cerebral disease.

The prognosis in acute rheumatism is also good as far as relates to the state of the affected joints; it is seldom that any very permanent injury is produced, unless the patient is otherwise out of health, and the rheumatic inflammation becomes the exciting cause of other serious and destructive action.

For an account of the after-changes which ensue from the cardiac affection, the reader is referred to the articles on Pericarditis, Endocarditis, &c.

TREATMENT OF ARTICULAR RHEUMATISM.—Numerous methods have been proposed for the treatment of articular rheumatism, and each has had its advocates; but, until recently, few have considered it feasible to leave such cases to nature, merely taking care that the patient be placed under favourable hygienic conditions. Many practitioners who would unhesitatingly adopt either a severely depleting or powerfully stimulating treatment, would shrink with alarm from the very idea of letting nature have her own way.

For several years I have treated some of my rheumatic patients in hospital on a purely expectant plan, and although the cases have not yet been tabulated, some of the results have been so decided as not to be mistaken. I am quite certain that many cases even of severe rheumatic fever get rapidly well without the administration of drugs, and on simply coloured or camphor water the improvement is often so quick and satisfactory, that had not the nature of the treatment been known, great virtue would surely have been ascribed to it; on the other hand, in many instances, the disease runs a lengthened course, with many partial relapses;—such tardiness is often found under other plans of treatment. In the Guy's Hospital Reports, 1865, are two communications containing the result of the treatment of forty-one cases of rheumatic fever, thirty-seven under Dr. Gull, and four under Dr. G. O. Rees, scarcely any medicine being given except mint water. Of the forty-one patients, twenty-two were males and nineteen females. The majority were under thirty years of age, only two above the age of forty years.

	Males.	Females.
The average number of days from the admission into the hospital to the cessation of pain; skin cool, and no relapse	13·1	15·5
The average number of days from the admission to complete convalescence; out of bed, and no relapse	16·6	21·4
The average number of days in hospital	27·6	26·8

The cases were all well-marked instances of rheumatic fever. In a large number of them the heart was implicated, and in such the duration of the symptoms was much longer: thus, the average duration of the acute symptoms in seven cases, in which there was no evidence of the heart being involved, was 8·5 days, but in six cases in which the heart was decidedly affected, 23·6 days.

The effect of the non-medicinal treatment on the supervention of heart disease could not be determined, as in the large majority of instances abnormal cardiac sounds were heard on admission.

Further trials of the expectant plan are much wanted, and every detail with regard to the condition of each patient should be carefully noted down and published, in order that the cases may be fairly analysed.

We will now give a short summary of the different methods which have been adopted, and have gained favour with the profession.

Venesection has been largely employed, and considered one of the most important curative agents in rheumatic fever. Sydenham was a strong advocate for this practice, and Cullen considered bloodletting to be the chief remedy in acute rheumatism, and thought blood ought to be drawn in large quantities, and the bleeding repeated in proportion to the frequency, fulness, and hardness of the pulse, and to the violence of the pain: he, however, thought that some bounds should be set to the bleeding; otherwise the recovery might prove slow. Bouillaud, in France, carried this plan to the fullest extent, and many English physicians during the present century have not been sparing of the lancet.

The condition of the blood in acute rheumatism, as shown by the buffy coat and cupped condition of the clot, evidently favoured the idea that bleeding was necessary in rheumatic fever, as in other forms of acute inflammation.

Venesection certainly gives speedy relief to the pains of the joints, even when practised to very small amounts, but free depletion tends to weaken the patient, and retard his restoration to health; it also causes the joint affection to linger, and favours relapses. Whether there is any real advantage in small depletions is questionable; I should certainly hesitate to bleed in the majority of instances, as there is no proof that it either shortens the duration of the joint affection, or lessens the tendency to cardiac complication.

At the time when bloodletting was commonly employed, some physicians were inclined to look upon it as of doubtful efficacy. Thus, Heberden states that, as far as he has been able to observe, the benefit of large and repeated bleedings is in most cases far from being clear and unquestionable.

Fordyce also remarks that, while it was the practice to remove the general inflammation by bleeding, metastasis frequently took place to internal organs and destroyed the patient, but that during fifteen years, when he entirely left off bleeding in acute rheumatism, he only lost two or three patients, although he treated several hundreds during that period. And, lastly, Dr. Willan states that he had observed that, by frequent bleeding, the pains, swellings, and febrile symptoms were not only aggravated at the time, but often protracted indefinitely, and that sometimes under such treatment the disease had lasted upwards of two months.

Mercurials, especially calomel, but a few years since were regarded as almost absolutely essential in the treatment of acute rheumatism, and even at present there are comparatively few who would venture to treat the cardiac complication without their aid: it is, therefore, important to decide the question of their utility.

If we compare the results obtained from cases treated freely by mercurials with those treated without mercury, it will be seen that, as far as the joint affection is concerned, the latter are decidedly more favourable than the former: such being the case, it seems difficult to understand why, if the inflammation of the heart's membranes be identical with that of the structures of the joints, mercurials should be of more value in the cardiac than in the articular disease.

Opium has usually been combined with calomel, partly to check the action of this drug upon the bowels, and partly to alleviate the painful affection of the joints. That opium in this disease is far more valuable than the calomel with which it is conjoined, is beyond doubt; it gives relief from the pain, promotes sleep, and prevents exhaustion of the nervous system; at the same time it encourages the true action of the skin, by increasing the capillary circulation.

A small dose of some preparation of opium, or of a salt of morphia, may generally be advantageously administered at night, and even once or twice a day, if the symptoms calling for its employment be urgent. Moderate doses of calomel may now and then be combined with opium, to promote the excretion of bile, and prevent constipation.

It is probable that opiates act merely as palliatives, and have no direct influence on the progress of the disease.

The use of *antimonial preparations*, more especially of tartar emetic, has been advocated in acute rheumatism; but experience seems to show that they have no power in checking the duration of the joint disease or of mitigating the cardiac affection; they are not much employed at the present time, except in very small doses, and then simply with the view of promoting the action of the skin.

Colchicum is a medicine which is still much used by a large number of practitioners, from the impression that gout and Rheumatism are affections closely allied to each other, and consequently that any drug valuable in the one disease must necessarily be useful in the other. Of the powers of colchicum in subduing true gouty inflammation there is no possible doubt; but in regard to acute rheumatism, the case is very different. As the result of my own experience, I may state, that colchicum possesses no influence in checking the progress of rheumatic fever; that when given in large doses, so as to lower the tone of the vascular system, it affords temporary ease, but not more than any other vascular depressant; and furthermore, that colchicum is a dangerous drug in many cases, as it acts as a direct cardiac sedative, and the chief danger to be feared in acute rheumatism is the loss of power of the heart.

Cinchona Bark and Quinine.—Although bark had been previously employed in the treatment of acute rheumatism, by Drs. Morton, Hulse, and Fothergill, Dr. Haygarth was the first who used it extensively, and brought forward clinical evidence of its utility; but the treatment did not appear to gain much favour with the profession; doubtless the large amount of cinchona bark necessary to produce a decided effect upon the system was a great drawback upon its administration: the substitution of quinine for the bark itself removed this objection, and the sulphate of the alkaloid has been tried very largely in France, and in very considerable doses, varying from one to six grammes in the twenty-four hours. From the statements of Briquet, Monneret, and Vinet, this remedy has a very decided influence upon the course of the fever, acting as a powerful sedative upon the circulation, and allaying the pain and swelling of the articulations; it often appears to prevent the development of cardiac complication, and even when this is present, it exercises no injurious influence. The effect of the quinine upon the disease is most decided when given in full doses, even to the extent of producing uncomfortable symptoms in the head and stomach.

Alkaline and Saline Treatment.—The treatment of acute rheumatism by saline and alkaline remedies has long found favour with the profession, and many modifications have been adopted; but before speaking of their relative merits, it will be well to define clearly what is meant by the saline and alkaline treatment.

There are certain saline remedies which, after absorption into the system, are eliminated by the kidneys in the same state as when they enter the stomach; for example: (1) nitrate of potash, chlorate of potash, and other salts in which the base is conjoined with a mineral acid; (2) alkaline salts with carbonic acid, in the form of the neutral or bicarbonate of the base; (3) salts with alkaline bases united with a vegetable acid, as citric or tartaric acid. Although these salts are neutral in reaction, when introduced into the stomach, they become speedily altered in the blood, the acid is decomposed, and a carbonate of the base appears in the urine; and hence, although they produce no alkaline effect upon the mucous membrane of the alimentary canal, yet upon the blood and the secretions their alkaline effect is well marked. Whether this effect on the blood is exactly the same as that of the free alkalies, or their carbonates, has not yet been determined.

Saline remedies have sometimes been employed in small doses, simply for the purpose of acting upon the secreting organs; at other times they have been given in very large doses, in order to alter the character of the blood itself, or powerfully influence the vascular system.

The first saline to be discussed is nitrate of potash. This remedy was used about a hundred years ago by Dr.. Brocklesby, in conjunction with bleeding. He ordered a dilute solution of the salt in water-gruel (about 120 grains to the quart), and as much as an ounce or more of nitre in the twenty-four hours. With this he states that he cured many cases in seven or eight days.

Since the above date it has been employed by M. Geudinn, M. Martin-Solon,[1] and, in this country, Dr. Basham.[2] From M. Martin-Solon's communication we cannot deduce the real value of the treatment, as the details are not sufficiently recorded, and it has been shown that under any treatment many cases get well in a comparatively few days.

Dr. Basham states that one, two, or even three ounces of the nitrate, freely diluted, may be taken in the twenty-four hours without inconvenience. He looked upon and employed the nitre as an adjunct to other remedies, and hence it is difficult to determine from his table of seventy-nine cases of acute rheumatism the real influence of the salt in combating the disease. Dr. Basham considers the local application of nitre of great value in causing abatement of the pain and swelling of the joints.

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[1] De l'Emploie du Nitrate de Potasse à haute Dose dans le Traitement du Rheumatisme articulaire. (Bulletin gén. de Thérap. août, septembre et octobre, 1843.)

[2] Med.-Chirur. Trans. vol, xxxii.

The nitre treatment, upon the whole, seems to have been followed by good results, and in Dr. Dickinson's record of seven cases treated in St. George's Hospital a favourable result was obtained.

Alkaline Treatment.—Although an apparently over-acid state of the body, and an increased amount of fibrin in the blood, in acute rheumatism, would naturally suggest the value of alkalies as remedies for this condition, it does not appear that they were systematically employed until the year 1847, when Dr. Wright published a communication on the subject. Since that period both Dr. Fuller and the writer have made a full trial of them, one administering the alkali combined with some vegetable acid, the other prescribing it in the form of the bicarbonate. Dr. Fuller's results will be found in his work on Rheumatism; the writer's earlier trials in a communication to the Medical and Chirurgical Society in 1855, in which an account is given of fifty-one consecutive cases, treated upon the full alkaline plan. The average duration of the disease in twenty males was 11·3 days, the duration under treatment 6·2 days; the average duration of the disease in thirty-one females was 15·7 days, the duration under treatment 7·3 days. In no case did any heart disease occur after the patient had taken the remedy forty-eight hours.

The plan consists in administering a dilute solution of bicarbonate of potash in about thirty-grain doses, every four hours, until the joint symptoms and febrile disturbance have completely disappeared. These doses produce no inconvenience either to the stomach or bowels; the urinary secretion is not notably increased, but its character is completely altered, and the reaction becomes either neutral or alkaline; it usually remains clear, but occasionally gives rise to a deposition of the triple phosphates. Upon the heart the alkaline bicarbonate acts as a sedative, reducing the frequency of the pulse sometimes forty-eight beats in the minute, but not causing any faintness.

When a patient is fully under the alkaline treatment, the blood is distinctly altered, and the coagulation of the fibrin takes place more slowly.

Many other remedies have been proposed for the cure of acute rheumatism, some of which it will be only necessary to enumerate: lemon juice has been prescribed by Dr. Perkins and Dr. Ciraud on the Continent, and in this country by Dr. G. O. Rees. It is usually given in quantities of from three to eight fluid ounces each day, and it is supposed to act as a sedative to the vascular system. It is doubtless a fact that, under its use, many cases of the disease rapidly get well, but, as has been shown above, this must not be taken as a proof of its curative power, and before we can decide upon its value, it will be necessary to have the tabulated results of a large number of trials of lemon juice, and to compare them with those obtained from other methods of treatment.

Other remedies used, either for the cure of the disease or the alleviation of certain of its symptoms, are, iodide of potassium, guaiacum, aconite, emetics, purgatives, diaphoretics, and hot-air baths.

Iodide of potassium is used to relieve the obstinate pains which linger when the acute symptoms have passed off, and more especially pains which are increased by heat, and most troublesome at night.

Guaiacum is valuable in sub-acute cases, when the circulation is weak, and the pains relieved by the application of warmth.

Aconite has been proposed to alleviate the acute pain of the joints, but when given in efficient doses it is apt to cause depression of the heart's action, and may be dangerous.

Hot-air baths exert a soothing influence, but great care is required if used during the time of febrile disturbance; and their curative value is somewhat questionable.

No proof has yet been given of the value of a free administration of emetics or purgatives; but proper attention must be paid to the state of the bowels.

Treatment of Heart, Lung, and Brain Complications in Acute Rheumatism.—Having given an account of several of the more important methods of treating rheumatic fever, it is desirable that we should inquire if any deviations are necessary when inflammation attacks the structures of the heart, lungs, or other internal organs. It has been already stated, that the articular inflammation leads to but little mischief, and that the joints rapidly recover their healthy state. It is, however, very different in the case of the heart; for there is a great disposition, both in the endocardial and pericardial serous membranes, to throw out lymph, which may lead to the thickening of the valves and adhesion of the surfaces of the pericardium: it is therefore a matter of no little moment to ascertain whether any plan can be adopted either to prevent such mischief supervening, or of rapidly and efficiently checking it if it has already taken place.

There appears to be every probability that the inflammation of the serous membranes of the heart is of the same kind as that of the joints, but it must be remembered that the structures themselves are of a somewhat different character, and remedies which produce little or no effect upon the joints may cause a decided action upon the cardiac tissues. It must also not be forgotten that inflammation lingers much longer in the heart, and is modified by the incessant movement of the organ.

It is a very common practice in cases of pericarditis, or even of endocarditis, to apply leeches over the cardiac region; and it is a clinical fact, that the tightness of the chest and pain are decidedly relieved by their application. Although it is difficult to explain the value of the local abstraction of blood in these cases, still I believe it is undoubted, and I cannot therefore hesitate to recommend it. The loss of blood need not be large, from three to twelve leeches are generally sufficient, and the bleeding should never be allowed to produce any appreciable weakening of the patient. Cupping may be employed in lieu of leeches, but I am inclined to prefer the slow loss of blood by the use of leeches to its more expeditious abstraction by the cupping-glass.

Blisters applied to the heart's region are also of much value, either before or after the application of leeches: care should be taken by previously applying collodion to the leech bites, and covering the surface of the plaster with tissue paper, to avoid the absorption of cantharidine, and the production of renal irritation. Now and then, if there be much effusion into the pericardium, the blister may be kept open, either with savine ointment, epispastic papers, or by some other means. When leeches have not been previously employed, the above precautions are less necessary. The simple application of a blister over the cardiac region is productive of much relief to the patient, and is followed by a decided improvement in both the heart's movements and sounds.

A very important question in the treatment of the cardiac complications relates to the value of mercurials. It has been shown that the mercurial treatment of the joint affection does not prevent the occurrence of inflammation of the heart, and it only remains to be ascertained whether this metal has any power of arresting inflammation after it has once ensued. *A priori*, it appears scarcely probable that a remedy, which has no influence in preventing inflammation, should have the power of arresting it when it has already commenced; but as the mercurial treatment is strongly advocated in such cases even by some who do not consider it as a preventative, it will be necessary to inquire a little further into the matter.

A few years since, almost every practitioner would have given this drug, and many at the present day scarcely dare omit its administration; for it is supposed to limit and control the inflammatory action, and to cause the absorption of the products which have been thrown out.

During the time of great febrile excitement, it is very difficult to get the system under the influence of mercury, and it may be that, when this is effected, it is rather due to the prior abatement of the inflammation than from the influence of the metal.

For many years I was in the constant habit of administering calomel in cases in which inflammation of the heart was present, but for the last eight or ten years I have not done so as frequently, and have seen no reason to regret the change of practice: the cardiac inflammation appears to have yielded quite as readily, and the patient, on the subsidence of the fever, has not had to suffer from ptyalism in addition to debility.

As the question cannot be considered in any way fully settled, it must be left to each practitioner to follow his own course, and form his own judgment on the value of the mercurial plan of treatment. When it is adopted, the common method is to give calomel in doses of from one half-grain to three or four grains, every four or six hours, usually combined with a small amount of opium, to prevent the purgative action of the mercurial. If much difficulty is experienced in producing the effect upon the gums, then inunction is often had recourse to, and this may be practised by causing a drachm or so of the blue ointment (unguentum hydrargyri) to be rubbed thoroughly into the skin of the inner side of the thigh, or into the axilla ; sometimes a blistered surface over the heart's region is dressed with the mercurial, instead of the green ointment. I have frequently adopted this latter method, and should prefer it, if mercurials are employed at all : it may be that the peculiar effect of the metal is produced upon the tissues in the neighbourhood of the blister, before the general system becomes affected by it.

During the whole course of treatment of the cardiac inflammation, the plan thought to be most advantageous for the joint affection should be steadily persevered with, as anything which favours the abatement of the systematic disease must also relieve the internal complications.

It is always a matter of the highest moment to insist upon the most complete quiet ; constant movement of the organ must necessarily take place, but everything should be shunned which increases this movement, or adds to the work which the heart has to perform. All chance of mental agitation, as well as bodily exertion, should therefore be strictly avoided.

After the inflammation has subsided, the heart is generally left in an irritable state : to allay this, a belladonna plaster is useful, and the administration of small doses of digitalis, combined with some salt of iron. More or less anæmia is always produced during the febrile disturbance, and this tends to keep up the excitement of the heart ; and hence the value of the ferruginous preparations.

If the pleuræ become inflamed during the progress of acute rheumatism, the affection may be treated upon the same principles as the pericarditis ; leeches and blisters may be prescribed, and calomel and opium given, if considered of value by the practitioner ; and the same remarks apply to the management of the brain and spinal complications which now and then arise.

Local Treatment in Acute Rheumatism.—In the majority of cases little or no local treatment is needed ; the affected joints should be protected by some light covering, as cotton-wool, flannel, or even a light handkerchief,—but nothing further is called for : but now and then the pain is so excessive, or the patient is so sensitive to it, as to render it desirable that some direct application should be made use of. Hot-water fomentations may be first tried, but if not found sufficient, belladonna may be added ;—a very convenient preparation for the purpose is the liniment of the British Pharmacopœia, which may be either sprinkled upon the hot flannel, or diluted with two or three times its bulk of hot water, and applied by means of a piece of lint, taking care to prevent evaporation by oil-silk or some other impermeable tissue. A solution of atropia, or atropia combined with morphia, forms a very clean and elegant substitute. The extracts of belladonna, henbane, conium, and aconite have been recommended, as also the tincture of opium and decoction of poppy-heads.

Alkalies and salines have also been extolled as topical remedies in acute rheumatism; Dr. Basham has employed a solution of nitrate of potash, and Dr. Fuller one of the carbonate of potash or soda. Leeches have sometimes been used to the inflamed joints: I have never seen occasion for their employment in genuine rheumatic fever.

Blisters applied so as to cover all the inflamed joints have been highly spoken of by Dr. Dechilly and others; Dr. Martin-Solon made a report on the subject to the Academy of Medicine in 1850, and in the discussion which ensued it was stated, that the treatment had no other effect than to quicken the subsidence of the inflammation in the joints. Dr. Herbert Davies has recently revived the use of free blistering, and orders armlets, wristlets, and even fingerlets of blister plaster, at the time when the inflammation is most acute; he recommends linseed-meal poultices to be subsequently applied, in order to promote the free flow of serum; he places these blisters entirely around the affected limbs, and, in the case of the knees, orders them of at least three inches wide, regarding any slight strangury which may arise as of little importance compared with the benefit afforded by the free vesication.

According to Dr. Davies, the blister treatment causes a speedy diminution in the frequency of the pulse, rapid subsidence of the joint affection, and lessens the liability to cardiac inflammation; within twenty-four hours after the removal of the blisters, the urine is stated to become alkaline in reaction. Dr. Davies' results in a large number of cases appear to be favourable.

Quino-alkaline Treatment.—During the last ten years, since the publication of his paper on the treatment of Rheumatism with large doses of bicarbonate of potash, the writer has made a very extensive use of the following plan, from which he thinks he has obtained more valuable results than from any other; it may be termed the quino-alkaline treatment, and is thus practised:—Sulphate of quinine is ordered to be rubbed up with a solution of bicarbonate of potash, to which a little mucilage and some aromatic, as tincture of cardamoms or spirit of chloroform, is subsequently added; each ounce-and-half dose contains five grains of the quinine and thirty grains of the potash salt, the quinine being reduced to the state of carbonate. To the adult the above dose is given each four hours, and persevered with until the joint affection and febrile disturbance have completely abated. When the quino-alkaline treatment was first made use of, a few days were allowed for the exhibition of the alkali alone, and then the quinine was added, but of late the quinine has been given from the first; it neither increases the thirst nor the furred state of the tongue, and its influence upon the heart is to lower its pulsations, but not to weaken them,—and hence, when peri- and endocarditis are present, its employment is not contra-indicated. If cardiac complications exist, local depletion and counter-irritation may be made use of.

In the sub-acute forms of articular rheumatism, the same plan may be employed, but in a milder form; that is, the doses may be smaller, or fewer in number.

If desired, the citrate of potash, or some other alkaline salt with a vegetable acid, may be substituted for the bicarbonate; when irritation of the intestinal canal is present, the bicarbonate appears to act as a valuable sedative, but when there is a tendency to constipation, the citrate or tartrate may be advantageously given, care being taken that absorption of the salt be not too much prevented by its action on the bowels.

The potassio-tartrate of iron may be added to the quino-alkaline draught after a time, and often with much advantage if any amount of anæmia is present.

Although the results obtained from a large number of trials have not yet been tabulated, the writer feels assured that the above treatment is much more efficacious than the simple alkaline plan, that there is far less tendency to the occurrence of relapses, and that the patient is left in a more satisfactory condition after the cessation of the febrile disturbance.

Diet and Regimen in Rheumatism.—In acute rheumatism, when the febrile disturbance runs high, food can only be advantageously given in the liquid form: it is, however, important to sustain the strength of the body as much as possible, and for this purpose milk, if it can be assimilated, is one of the best forms of nourishment, as it contains all the elements necessary for repairing the waste of the system. If milk in an undiluted state is too heavy, it may be mixed with an equal measure of soda water. Besides milk, beef-tea, mutton and other meat broths, and jellies, may be given. It is desirable also to introduce a certain amount of amylaceous and saccharine matters into the system; hence arrowroot, made with water, milk, or beef-tea, may be administered. Soda water, lemonade made with cream of tartar or citrate of potash, and flavoured with lemon, toast and water, or plain water, may be employed to allay thirst. Alcoholic stimulants are seldom needed in the young subject, unless there is great depression from the presence of serious cardiac mischief: wine, in true Rheumatism, has no tendency to keep up the specific inflammatory action.

As the fever abates, and the power of the digestive organs returns, the diet may be improved; at first bread, light puddings, then white fish, fowl, and afterwards ordinary meat; but care must be taken that the stomach be not distended nor the system disturbed by the presence of food incapable of being digested. Throughout the whole course of the treatment of Rheumatism, both in its acute and sub-acute varieties, the great object should be to sustain as much as possible the strength of the patient, for by this means the duration of the disease is diminished, and the subsequent recovery rendered less tedious.

During the height of the malady, when the skin is freely perspiring, care should be taken not to allow the surface to be suddenly cooled; for although the free action of the skin does not appear to give relief to the pains, yet a sudden check to it cannot fail to be injurious, and may lead to metastatic action.

Individuals who have once suffered from rheumatic fever should be extremely careful as to their clothing; they should always be completely clad in flannel, which may vary in thickness at different times of the year; the feet should be kept warm, and every precaution taken to avoid a chill.

B. MUSCULAR RHEUMATISM.

DEFINITION.—An affection of the voluntary muscles, of an inflammatory nature (?), but unaccompanied with swelling, heat, redness, or febrile disturbance.

SYNONYMS.—Designated according to the situation of the affected muscles, Lumbago, Torticollis, Pleurodynia, &c.; termed also Myositis, Myo-rheumatism, and Myodynia.

HISTORY.—By British writers this disease is often included, with other affections, under chronic rheumatism, a name manifestly incorrect, as the malady often assumes an acute character. On the Continent some authors have claimed for it a separate existence, and the disease can scarcely be looked upon as a chronic form of articular rheumatism, seeing that it is probable that its pathology is different.

DESCRIPTION OF MUSCULAR RHEUMATISM.—Muscular rheumatism usually commences as an acute disease, but has a considerable tendency to assume a chronic form; it may affect any of the voluntary muscles, but is prone to attack certain sets rather than others. The seizures are frequently sudden; sometimes, for example, a patient awakes in the morning and finds himself incapable of turning in bed or of twisting the neck, and the attempt to do so gives exquisite pain. If the affection is very acute, he may suffer pain even when quiet, for the muscles are often involuntarily thrown into a state of spasm; in the less severe forms the patient may be comparatively comfortable when at rest, but on the least movement he experiences agony by the paroxysm thereby excited. On examining the seat of suffering, no external phenomena are visible, but there may be slight tenderness on pressure; there is also a freedom from febrile excitement, at least at the onset of an attack, but as it progresses, thirst, loss of appetite, and heat of skin may ensue, probably due simply to the continuance of the pain, and the loss of sleep thereby occasioned. In muscular rheumatism the pulse is but little affected, and the urinary secretion preserves its normal state. A very important feature in this disease is the absence of inflammation of the heart, so characteristic of the articular affection. In the acute stages the symptoms increase towards evening, and are augmented by heat; but when the disease becomes chronic, the pain is not unfrequently relieved by its application.

The duration of acute muscular rheumatism is generally short, usually not more than a few days, seldom exceeding a week; but when its intensity has become mitigated, it often proves tedious, and may be prolonged for an indefinite period of time; it is also apt to be again lighted up, if the patient be exposed to any of its exciting causes.

The principal varieties of muscular rheumatism are as follows:—

Lumbago (lumbodynia), when the large masses of muscles on each side of the spine in the lumbar region are implicated. When acute, it renders the patient utterly helpless; the most intense agony is induced on the slightest attempt to rise in bed, or even to turn in any direction.

Torticollis (cervicodynia, or crick in the neck) is a term applied when the muscles on one side of the neck are affected. The patient is compelled to hold his head awry, in order to relax the muscles. Sometimes the sterno-mastoid is principally implicated, sometimes the disease extends to the muscles at the back of the neck.

When some of the intercostal muscles are the seat of the malady, the affection is called *Intercostal Rheumatism* (pleurodynia): the symptoms are pain in some part of the chest, rendered intense by the act of breathing, but relieved by such pressure as prevents the movement of the ribs.

The above three names are in common use, but many others have been occasionally employed by authors desirous of designating diseases simply from their locality; and thus have arisen the terms Scapulodynia, Dorsodynia, Cephalodynia, according as the muscles about the shoulders, or of the dorsal region of the spine, or those of the head, are particularly implicated.

As any of the voluntary muscles are capable of taking on morbid action, these names might be multiplied indefinitely; the walls of the abdomen, the muscles of the limbs, the tongue, pharynx, diaphragm, are stated to be sometimes implicated; and even some of the involuntary muscles, as of the œsophagus, stomach, intestines, and uterus, appear to be susceptible of the disease.

CAUSES OF MUSCULAR RHEUMATISM.—These may be divided into, 1st, those dependent on the peculiarities of the individual, and, 2d, those which are altogether external.

Causes dependent on the Individual.—True muscular rheumatism is seldom seen in very young subjects; it generally occurs after the age of puberty, and more commonly in full adult age.

One attack of the disease engenders a liability to its return, as is the case with articular rheumatism.

Individuals of a gouty habit are frequently attacked with a muscular affection, which cannot well be distinguished from ordinary muscular rheumatism.

Causes external and independent of the Individual.—The only external causes of muscular rheumatism appear to be cold and damp, more especially if combined with an over-use of the muscles : lumbago, for example, is often produced by straining the lumbar region ; and cold draughts of air, as from a partially open window, will frequently prove the exciting cause of an attack of torticollis.

PATHOLOGY OF MUSCULAR RHEUMATISM.—The nature of muscular rheumatism is usually regarded as closely allied to articular rheumatism, the difference in the symptoms being supposed to depend on the peculiarities of the structures which are affected in the two diseases. Some reasonable doubt may, however, be entertained of the correctness of this opinion, for the following reasons :—If the proximate cause of the muscular affection is the same as that of the articular, the heart would probably become inflamed in a certain proportion of the cases ; but it is not so : and, again, there is an absence of the peculiar secretion from the skin, so marked in articular rheumatism. The state of the blood has not yet been ascertained, but there is reason for believing that the fibrin is not much augmented.

For the present we must be content to await the result of more numerous and searching inquiries, before attempting to explain the true nature of muscular rheumatism.

DIAGNOSIS OF MUSCULAR RHEUMATISM.—Acute intercostal rheumatism may be mistaken either for pleurisy, or for a neuralgic affection so frequent in hysterical subjects : from the former it may be separated by the absence of the friction sound and other signs of inflammation of the pleura ; from the latter, by the absence of well-marked spinal tenderness. It is sometimes difficult to distinguish the chronic form of muscular rheumatism from the dull pains arising from other causes, as from progressive muscular atrophy, the presence of metallic poisons in the system—especially lead and mercury—and likewise the poison of syphilis ; also from ordinary inflammation of the muscular tissue. Lumbago may be mistaken for a painful affection of the loins, arising from calculi or gravel in the kidneys.

A careful examination of the history is usually sufficient, in each instance, to enable us to arrive at a correct diagnosis.

PROGNOSIS OF MUSCULAR RHEUMATISM.—Muscular rheumatism, although difficult to cure, is not attended with serious results ; there is not the fear of cardiac inflammation, the great source of danger in articular rheumatism. The sciatica arising from lumbago is often both painful and obstinate.

TREATMENT OF MUSCULAR RHEUMATISM.—In the acute stages of muscular rheumatism, especially in lumbago, local depletion, generally by cupping, is resorted to. That this remedy gives temporary relief, there cannot be a doubt, but whether it be necessary or not is another question. My own opinion is, that it is seldom or never called for, and that the same relief may be obtained from other topical applications which are less open to objection. Hot fomentations are very valuable; as, for example, spongio-piline, wrung out of hot water, and applied either alone or sprinkled with tincture of opium, or the liniment of belladonna, or a combination of the two. When the affection becomes more chronic, then a thinner piline lightly sprinkled with belladonna liniment, with or without the addition of the volatile oil of mustard, proves very useful, acting as a counter-irritant, and at the same time relieving pain by the anodyne properties of the belladonna.

Turpentine fomentations, or stupes, may be substituted in some instances for the above applications.

Occasionally blisters are applied, and if the affection prove obstinate, they may be kept open, with the addition of morphia or some other anodyne.

The internal remedies which have been employed in cases of muscular rheumatism are numerous. When the affection is acute and recent, benefit often arises from the use of salines, which act on the skin and kidneys, and alter the state of the blood. From my own experience I should recommend the acetate of ammonia in large doses, combined with some alkaline salt, as the bicarbonate of potash, and with the addition of iodide of potassium. The time, however, soon arrives for the use of some medicinal agent, as quinine, which exerts a marked action upon the nervous system; and this may be advantageously combined with the other remedies. Sometimes large doses of the hydrochlorate of ammonia are useful; and in cases which occur in gouty habits, colchicum alone, or in combination with other medicines, may be prescribed with much benefit.

When the disease becomes chronic, medicines which increase the capillary circulation are generally useful: guaiacum stands at the head of the list, and may be given either in substance or as the ammoniated tincture; after guaiacum follow sassafras, mezereon, turpentines, resins, and balsams.

Sulphur in small doses is frequently of much advantage, and it can be administered in substance or in the form of the sulphur waters of Aix-la-Chapelle, of Aix in Savoy, or Barèges.

Arsenic is likewise occasionally adopted as a remedy in long-standing, obstinate cases.

When the pain has subsided, friction and electricity are often resorted to, in order to diminish the stiffness and to restore tone and activity to the muscles.

The diet should be generous, with a moderate amount of stimulants; the patient should be warmly clad in flannel, and every precaution taken to avoid chills.

GONORRHŒAL RHEUMATISM.

By Bernard Edward Brodhurst, F.R.C.S.

The affection which is known as Gonorrhœal Rheumatism consists of inflammation of and about joints, following upon urethral irritation, and preceded for the most part by a muco-purulent urethral discharge.

Swediaur and Monteggria are said to have described this affection, and to have published cases illustrative of the disease; but there is not anything to be found in their writings which entitles them to the merit which has been claimed for them; and indeed there ought to be no hesitation in saying that the disease does not seem to have been recognised until Sir Astley Cooper mentioned it in his lectures. Mr. South says: "Gonorrhœal Rheumatism and gonorrhœal ophthalmia were, I believe, first mentioned publicly by Astley Cooper, and the first of these affections he considers is not an infrequent disease; but it appears to have been previously observed by the elder Cline, for, in reply to the question put to him by Cooper, whether he had ever seen rheumatism produced from gonorrhœa, he said, 'Several times.'"[1]

Sir Benjamin Brodie has given some pathological account of this disease, and having described its nature, he continues thus:—"The disease is usually described under the name of Gonorrhœal Rheumatism, though it is plain, from the course of its symptoms and from the effects of remedies, that it differs from ordinary rheumatism in many essential circumstances, and though there seems to be no doubt that, while it occurs in most instances as a consequence of gonorrhœa, it may take place quite independently of gonorrhœal infection."[2]

Sir Astley Cooper relates the following case:—"An American gentleman came to me with a gonorrhœa, and after he had told me his story, I smiled, and said, 'Do so and so,' particularizing the treatment, and that he would soon be better; but he stopped me, and said, 'Not so fast, sir; a gonorrhœa with me is not to be made so light of; it is no trifle; for in a short time you will find me with inflammation in the eyes, and in a few days after I shall have rheumatism in the joints. I do not say this from the experience of one gonorrhœa only, but from that of two, and on each occasion I was afflicted in this manner.' I begged him to be careful to prevent any gonorrhœal matter coming in contact with the eye, which he said he would. Three days after this I called on him, and he said, 'Now you observe what I told you a day or two ago is true.' He had a green shade on, and there was ophthalmia of each eye. In three days more he sent for me rather earlier than usual for a pain in his left knee; it was stiff and inflamed. I ordered some applications, and soon after the right knee became affected in a similar manner. The ophthalmia was with great difficulty cured, and the rheumatism continued many weeks afterwards."

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[1] A System of Surgery, by Chelius, translated by John F. South, vol. i. p. 217.

[2] Pathological and Surgical Observations on the Diseases of the Joints. 5th edit. p. 43.

That which is here so graphically related may be observed in almost every instance of Gonorrhœal Rheumatism; and, with the exception of the ophthalmia, it is exactly the manner in which every first attack of Gonorrhœal Rheumatism arises and proceeds. Gonorrhœal ophthalmia is much less frequently observed than gonorrhœal articular rheumatism: probably it does not occur more than once in a dozen instances of Gonorrhœal Rheumatism.

Gonorrhœa then being established, one or more joints become, in the course of from ten days to three weeks, stiff, painful, and swollen, the patient having, perhaps, exposed himself to the weather, sitting or walking in wet clothes, or to a draught of cold air. At the same time the feet may be painful and the conjunctivæ inflamed; there will be considerable fever, with dry skin and a furred tongue. Probably, as the articular inflammation increases, the urethral discharge will diminish; again it will become more abundant, and at length cease or degenerate into a gleet.

The first attack of Gonorrhœal Rheumatism is invariably preceded by a specific gonorrhœal discharge; a subsequent attack may be preceded by urethral discharge which is not of a specific character; and, also, the same character of articular disease may be re-excited without the urethral discharge being developed.

Both the robust and the debilitated suffer from this disease, but they suffer differently. The fever and inflammation are proportionate to the plethora which may exist. In the young and plethoric the inflammation is of an acute character, and lymph is for the most part deposited on the synovial membranes, giving rise to false ankylosis; whereas, in the debilitated, serum will probably alone be effused. In both cases the joints are liable to be destroyed; in the former through the deposit of lymph and the production of false ankylosis, and in the latter through the destruction of cartilage.

The joint having become inflamed, a large effusion of serum takes place into the synovial cavity; but, although there may be great tension, suppuration never occurs. Absorption of the effused serum takes place, and the joint may resume its healthy action. The limb remains in a semi-flexed position during the period of effusion into the joint; for in the flexed position of the limb the surrounding structures are somewhat relaxed, and consequently they yield to the bulging membrane with its contained fluid. When, however, the hip is inflamed, the limb remains much more extended than in ordinary hip-joint disease.

The knee is more frequently affected than any other joint, being a large and complicated joint, and less protected by muscle from atmospheric influence.

After the first attack of this disease, the patient is exceedingly liable to a recurrence of it. Usually the second attack is, as the first, the result of gonorrhœa and exposure to cold and wet. But, although Gonorrhœal Rheumatism will now almost certainly be excited by a specific discharge, any urethral discharge or urethral irritation would seem to be sufficient to re-excite the disease when the patient has once suffered from it.

On this second occasion probably the inflammation will be less, but the joints will be longer in recovering mobility, and one or more will possibly remain ankylosed. Sir Benjamin Brodie relates the case of a patient who suffered from four attacks of this disease in the course of some few years, in whom he says that "inflammation of the urethra was in all of them the first symptom, which was followed by purulent ophthalmia, and afterwards by inflammation of the synovial membranes and swelling of nearly all the joints. In two of these attacks he attributed the discharge from the urethra to his having received the infection of gonorrhœa; and in the two others to the use of the bougie."[1]

Slight stiffness may remain for several weeks, and a crackling sensation will probably be communicated to the hand on moving the limb; but this may also at length cease, and the joint will resume its normal condition both in regard of size and motion.

Each attack is more virulent in its character than the preceding, and in proportion to the debility of the patient. The female seldom suffers from this disease, but whenever I have observed an instance, it has always terminated in ankylosis.

When gonorrhœal ophthalmia occurs, the conjunctiva, sclerotic, and iris may all become affected. It is not for the most part a severe form of ophthalmia, and it readily yields to treatment.

Although the effusion into a joint may be very considerable, dislocation never occurs, and in this point the disease differs widely from ordinary rheumatism, where the tendency is for the articular surfaces to become more or less displaced; but in this disease ankylosis is induced rather than dislocation.

In no case that I have met with has there been so much effusion into the joints, and so much pain, as in the following instance; neither have I met with a case where the patient enjoyed such redundant health. A dark-haired young man, who was attached as lieutenant to one of our regiments serving in a tropical climate, had contracted gonorrhœa, and having laid himself down on a low broad wall he fell asleep, and so remained for some hours, and until after sunset. He awoke in great pain, and, as it seemed to him, this pain was spread all over the body, but especially it was felt in one hip: here, however, the pain was so acute that it was with difficulty he could be removed to bed. Besides the hip, the shoulders, knee, and ankle became affected; but in none of these joints was the inflammation so acute as in the hip, and they all passed through this inflammatory condition without material injury, and recovered perfectly. In the hip the effusion was so great that it was thought suppuration must take place: the swelling, however, subsided at length, leaving the joint stiff and immovable. Until this effusion was removed he suffered an agony of pain.

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[1] Op. cit.

Occasionally the urethral discharge alternates with articular inflammation, or it degenerates into a gleety discharge, while the articular inflammation assumes a chronic character. Thus, a young Jew, who had contracted gonorrhœa, was soon after seized with pain in several joints. The urethral discharge ceased as the articular inflammation became developed, and it recurred as the pain in the joints was removed. This continued for several weeks, until on a winter's evening he was exposed to cold, when, an access of inflammation occurring, he found during the night a finger-joint excessively painful and somewhat swollen. The joint became ankylosed in the course of some few days. All the other joints recovered well. The gleet remained for many weeks.

The articular inflammation appears to be of a more injurious character when a second or third attack of Gonorrhœal Rheumatism occurs. The first attack may leave no trace behind; but a subsequent attack seldom fails to do so: it may cripple the patient for life, as happened in the following case of a young man, nineteen years of age, of a somewhat strumous diathesis. In this patient gonorrhœa appeared on the seventh day after infection. In the course of a fortnight the knees became painful and swollen, and they continued in this state for about two months, when the inflammatory condition subsided, and the joints resumed their normal condition. Two months after this first attack had disappeared, he was again infected with gonorrhœa. In ten days several joints became inflamed, as the right temporo-maxillary articulation, as well as the hips, the knees, the ankles, the shoulders, the elbows, and the thumbs. The upper limbs and the ankles recovered well and regained their mobility, but the hips, knees, and the jaw became ankylosed.

The following is a remarkable case, in which, after several attacks of Gonorrhœal Rheumatism, the disease was again set up without urethral discharge appearing. A gentleman, twenty-five years of age, acquired gonorrhœa, which was soon followed by pain and swelling of the knees. The urethral discharge appeared on the seventh day, and some few days later synovitis supervened with great effusion into the knee-joints. The skin was hot and dry, and he suffered acutely, so that every movement was attended with much pain. The urethral discharge continued for two months, and then ceased entirely, and the swelling and stiffness of the knees also at length disappeared, having lasted for three months. After another interval of three months this individual was again affected with gonorrhœa. The urethral discharge again appeared on the seventh day, and it continued for two months. After some few days the left hip-joint became inflamed, as well as the ankle and tarsal joints. This attack was of a much more severe character than the former one—the effusion was greater and the pain more acute. After a period of ten months my patient was able to walk with the help of sticks. Stiffness and a painful condition of the limbs continued yet, however, for many months; but at length he regained the use of his limbs. After a lapse of several months, again he contracted gonorrhœa. On this occasion the symptoms closely resembled those which occurred on former occasions; but he never entirely recovered from the stiffness which resulted from this attack of articular inflammation. Now, both hips, both ankles, and one knee became inflamed, and he also suffered from ophthalmia. Ankylosis did not occur, but there was a certain amount of stiffness of the joints which prevented him from rising after he had been seated for some hours. About six months after he was able to walk about, he married. Painful attempts were made to consummate the marriage, but it was found to be impossible: Dr. Lever discovered that occlusion of the vagina had taken place. At this time, namely within a very short period of marriage, articular inflammation recurred. There was now, however, no urethral discharge whatever. But the articular inflammation proceeded, and at length produced ankylosis of every joint in succession; so that in five years the whole skeleton was implicated: the atlas was ankylosed together with the axis, and in consequence the head could not be moved; and all the vertebræ were ankylosed together; and the hips, knees, ankles, shoulders, elbows, wrists, and jaw were so firmly fixed that no movement whatever could be obtained.

An interesting example of this disease has been for some time under treatment in St. George's Hospital, of which the following are the principal points. Edward G——, aged 46, having been treated as an out-patient by Dr. William Ogle for some time, was at length admitted into Fitzwilliam Ward, on acount of effusion into and great thickening about the knee-joint. The left wrist was firmly ankylosed, and the spine in its entire length was motionless—ankylosis of all the vertebræ having taken place. The ribs also were ankylosed, and the breathing in consequence was purely diaphragmatic.

In the year 1855 (thirteen years prior to his second admission), he was a patient of Dr. Wilson's, in Cambridge Ward, for Gonorrhœal Rheumatism.

Three months before he was admitted into the hospital, he was exposed in the hunting-field, being a groom, to wet and cold, at the same time that he was suffering from an attack of gonorrhœa. He was admitted with pain in the feet and shoulders, in the right elbow, and over the clavicles; but at this time the gonorrhœal discharge had stopped.

Since that period he has never suffered from gonorrhœa; but he has had pain in various joints, and gradually his back and neck have become stiff, as well as the left wrist and the right knee. He was scarcely aware that any morbid process was going on in the spine, for there was little or no pain, and during the whole of the time between his discharge from the hospital and his re-admission he was able to perform his duty as a coachman.

Treatment.—When Gonorrhœal Rheumatism is vigorously treated in the commencement of the attack, the joints may become affected in a slight degree only. If there be much constitutional disturbance and inflammatory action, purgatives may be exhibited, and a small quantity of blood may be taken from the arm; while local fomentations may be employed, and a splint of gutta-percha to keep the affected joint perfectly at rest. After venesection, a full dose of opium gives great relief, and if it is administered with ipecacuanha, as in Dover's powder, the secretion of the skin is increased. The sweating which is thus produced is beneficial; but this increased action of the skin is best promoted in the Turkish bath. I have known the pain about the joints to cease entirely in the bath. Some time since, I saw a gentleman who suffered very acutely from pain and inflammation consequent on this form of disease of the joints, and in whom the tension from effusion was also excessive. He was lodged by my desire in a house attached to a Turkish bath, and each day he was carried down into the bath. When profuse perspiration was obtained, the pain left him; and absorption of the fluid within the synovial capsules was certainly promoted by exposing the body to the high temperature of the bath.

In an acute attack, abstinence from flesh meat, as well as from fermented and distilled liquors, is absolutely necessary.

When such measures are promptly taken, an acute attack of Gonorrhœal Rheumatism may generally be cut short. When, however, inflammation tends to become chronic, iodide of potassium may be given with advantage: thirty to forty grains being administered daily. It is preferable to abstract a small quantity of blood from a vein than to apply leeches to the inflamed joints: leeches not unfrequently aggravate some of the symptoms, and induce also suppuration in the cellular tissue. For these reasons, any benefit to be derived from their use is doubtful, and at times they certainly increase the evil they are intended to mitigate.

After the first or second attack, or when the patient is debilitated the treatment should be of a slightly stimulating and tonic character:

depletion will aggravate all the symptoms, and increase the effusion. Opium may be given freely, and iodide of potassium in small doses. Gutta-percha splints should always be used during the period of effusion, to prevent motion.

So soon as pain and swelling have ceased, gentle frictions with shampooing should be employed to restore mobility. Much time will probably be required to effect this object; and it may be necessary, if adhesions have formed, to flex the limbs forcibly after chloroform has been inhaled. In many cases mobility may be restored, even after very firm adhesions have been formed.

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